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REPORT OF THE ALDRIN/DIELDRIN ADVISORY COMMITTEE TO WILLIAM D. RUCKELSHAUS, ADMINISTRATOR, ENVIRONMENTAL PROTECTION AGENCY

Environmental Protection Agency
Washington, D. C.

28 March 1972

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REPORT OF THE ALDRIN/DIELDRIN ADVISORY COMMITTEE*

TO

WILLIAM D. RUCKELSHAUS, ADMINISTRATOR
ENVIRONMENTAL PROTECTION AGENCY

March 28, 1972

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*Established Under Provisions of Section 4.c of the Federal Insecticide, Fungicide, and Rodenticide Act.

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March 24, 1972

Dear Mr. Ruckelshaus:

The report of your advisory committee on aldrin and dieldrin is enclosed. The report consists of first, a brief review of our analysis of the situation, in which we have responded to the charge to the committee; second, a series of six recommendations; third, a set of six technical appendices, each of which reviews the pertinent scientific literature in a given area, and upon which most of our analysis is based.

The committee had 4 day-long meetings in Washington. All of the individuals or groups who wished to appear before us did so, and in addition we received verbal and written testimony from several authorities whose advice we solicited, on subjects as diverse as chemical carcinogenesis and agricultural economics. In addition, each committee member performed, at his own institution, extensive research and review in the area of his own expertise, and had the benefit of the knowledge and views of appropriate colleagues at that institution.

I am happy to tell you that the committee was unanimous in the views and recommendations we bring to you, and that although our technical backgrounds are so diverse, we readily found agreement both upon the general approach we recommend to you, and upon the specifics.

Yours sincerely,

R. D. O'Brien, Chairman
Advisory Committee on
Aldrin and Dieldrin

Enclosure
RDO'B: o

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REPORT OF THE ADVISORY COMMITTEE ON

ALDRIN AND DIELDRIN 3/23/72

Aldrin and dieldrin are compounds used widely today for a variety of insecticidal purposes. Their major use is for control of soil insects in corn. Aldrin is readily converted to dieldrin in soil and many biological systems, and thereafter is only slowly degraded to biologically inactive products; as a result dieldrin has contaminated the American diet in a widespread way, although the contaminating amounts are very small. Several reports claim that the world's environment is generally contaminated with one or both compounds, but these claims must be regarded with caution because few studies have used analytical methods which respond only to these compounds. For example, sulfur and PCB's (polychlorinated biphenyls) may be mistaken for aldrin. A compelling demonstration of the need for unusually careful analysis is the report that standard assays for aldrin upon soil held in sealed storage since 1909 (long before aldrin or dieldrin were in use) showed apparent aldrin and dieldrin residues in 20 of the 34 samples assayed.

Human intakes and their significance.

There is clear evidence that Americans consume significant amounts of aldrin and dieldrin daily; for 1968-9, for instance, the average intake for a 70 kg adult was 5 ug/day. Levels in dairy products were highest (0.05 ppm of food), in meat products a little less (0.03 ppm) and in cereals and vegetables quite small

(below 0.01 ppm). It should be noted that these intakes do not accumulate in the body indefinitely; instead the levels in the body achieve a fixed concentration characteristic of the intake level. In the United States the levels in the body fat of the general population average 0.15 ppm. This raises the questions: are these fat levels a cause for alarm, and can steps be taken to reduce or eliminate them without disproportionate damage to the national welfare?

We consider that the available evidence suggests that the small human intakes and consequent body burdens are harmless. Not only are they far below the amounts shown to be toxic to laboratory animals, but human beings, either volunteers or those exposed industrially, have taken in much larger amounts than the general population with no untoward results. For example, occupationally exposed men received 50x the average intakes of dieldrin for periods of from 4 to 13 years, and not only showed no sign of toxicity, but failed to show the minor effects upon liver function which are early signs of bodily changes produced by such compounds. Supporting evidence is that we have a series of depositions from University Extension Agents, pest control applicators and retailers testifying that no untoward effects have been reported from the use of aldrin or dieldrin.

But there is no doubt that dieldrin can cause malignant tumors of the liver in one strain of mice. In one other strain, hyperplastic nodules of the liver were produced, but no carcinomas.

Consequently one cannot dismiss the possibility that sufficient doses of dieldrin could be carcinogenic in man, but the degree of certainty is inadequate to require prompt elimination from the diet. One must also note that in rats, dieldrin at 20 ppm or more in the diet decreased malignant mammary and lymphatic tumors; for instance, in males 14% of controls had such tumors, but only 6% of treated animals had them.

It seems clear that the species selected for test is important. Furthermore, there is reason to believe that fetal organisms, because of their high mitotic rate, might be unusually sensitive, and it is clear that placental transfer occurs in humans(271). Consequently we would like to see studies on effects upon fetal animals whose mothers are treated and which are also postnatally exposed. We shall propose, below, tests to improve our ability to estimate human hazard, as well as measures which we believe will reduce human dietary intakes.

Effects on wildlife and plants.

Data upon the toxicity of aldrin and dieldrin to fish and crustaceans lead us to accept such terrible accounts as that describing the effects of 1 lb/acre of dieldrin on 2000 acres of Florida salt marsh used for sandfly control: "--fish kill was substantially complete. The minimum immediate overall kill--- was 20-30 tons of fishes ---. Crustaceans were virtually exterminated throughout the area"(105). For such reasons, we believe that applications to aquatic habitats must be forbidden; one

should recall (see appendix) that even 3 ppb in water can cause measurable toxic effects in some fish.

As for effects upon terrestrial vertebrates, the hazards are less extreme. Thus rather high levels of dieldrin (ca. 1 ppm in the diet) are needed for production of thin-shelled eggs in ducks, in one of the few carefully controlled experiments(162). In sparrow hawks, the high dose of 3 ppm of dieldrin plus 15 ppm of DDT produced a maximum eggshell thinning of 16%(202). Furthermore the reported effects of dieldrin upon carbonic anhydrase (thought to be causal in eggshell thinning) have recently been shown to be artefactual, being caused by coprecipitation of the soluble form of the enzyme used in laboratory studies(201). Much of the data on effects upon wild birds is impossible to evaluate because dead birds were collected without establishing the cause of death, and they contained a variety of pesticide residues. Data upon bald eagle deaths is particularly suspect in this regard(191).

Nevertheless we accept that in at least some avian species, quite low levels of aldrin or dieldrin may have adverse effects; thus 1 ppm of aldrin in the diet reduced egg production of pheasants by 17%, in quail by 23% and 1 ppm of aldrin or dieldrin was lethal to 100% of quail chicks(56). To avoid adverse effects on wildlife, aldrin and dieldrin must be used in ways which cannot lead to intake levels of several ppm for birds. The use of aldrin applied directly to soil at 1 lb/acre over 16 years leads to levels of about 1 ppm (aldrin plus dieldrin) in a variety of insects, and

less than 0.02 ppm in a variety of seeds of plants grown in that soil(155). Such usage is unlikely to lead to substantial effects on wildlife.

There appear to be no reports that aldrin or dieldrin have adverse effects upon plant life.

Aldrin and dieldrin in sediments.

Because use of aldrin in soils is so extensive, special attention needs to be given to the question of loss by means of sediment run-off from the soils to which aldrin is applied and firmly bound. The 1971 study of Rykman et al. for EPA points to this as the dominant route for contamination. It points to the millions of tons of sediment carried in the country's major rivers, and provides one estimate that, as an example, "about 10 tons of dieldrin are annually carried in the Mississippi at St. Louis"(p.15). If these data were reliable, we would have to agree that their source of pollution was exceptionally important and would perhaps suggest that soil application of these agents should be banned. But this estimate assumed that the residues in farm soils and in river sediments are the same. On p.14 this same report states that for Iowa (the only state for which both funds of data are given) the dieldrin in farm soils averages 107 ppb, but in river sediments averages 11 ppb, and they compute that "if this amount of dieldrin was in the sediment load passing St. Louis, the total annual dieldrin pesticide load would be 260 lbs"; thus the alarming 10-ton figure is utterly misleading.

In addition, it is not at all clear what is the toxic significance of these sediments. We do not know if the dieldrin or aldrin which is bound to these sediments is biologically available. If sufficiently firmly bound, it might be able to pass through the tracts of bottom-feeding fish without being absorbed and in fact the data of Appendix VI-A suggest that bottom-feeding fish do not acquire especially large residues. By contrast we may assume the water levels of dieldrin to be fully available biologically; dieldrin (but not aldrin) is indeed widespread in river water, but the highest recorded level (in 1968, in the Tombigbee River, Columbus, Miss.) was 0.4 ppb; typical Mississippi River levels are about 0.015 ppb. The EPA suggests 10 ppb as acceptable water qualtiy criteria (although data in Appendix II show that even 3 ppb can have undesirable effects in some fish). The amounts in river water do not seem to be alarming.

Economic considerations.

One of the problems we have encountered is the grave inadequacy of the estimates of the economic implications of insecticide use. Let us illustrate this with the major economic usage; aldrin for treatment of soil insects in corn. Some problems are:

(a) There is little experimental data to show the effects upon yield of withdrawing aldrin or substituting it with non-persistent compounds.

(b) The available estimates deal with average effects on national yields. But in fact these will involve very drastic reductions in limited areas of severe infestations, with lesser

effects elsewhere. How much consideration should we give to the impact upon individual farmers in such cases?

(c) The importance of insecticide withdrawal is likely to vary from year to year, as pest infestation varies.

(d) Withdrawal effects may seem small at first, but become progressively worse as the residues of aldrin or dieldrin dwindle over the years.

(e) The implications of a given dollar loss, either in reduced yield or increased treatment cost, depends on the margin of profit the farmer operates on, and is hard to assess. Furthermore, the profit depends largely upon such artificial factors as the support prices in any year, and the status of the export market.

In spite of these grave difficulties, rough estimates have been made by Dr. J. H. Berry of USDA, suggesting that the discontinuance of aldrin and dieldrin would lead to losses (at 1969 prices) of $14 million for crops where no replacement is possible, plus $34 million for crops where alternative insecticides are available (this includes aldrin use on corn), for a total of $48 million (these estimates assume no price increases for the crop; clearly the losses could go to zero for the producer, if all extra costs were passed along to the consumer). It should be emphasized that although we have no quarrel with the <u>procedures</u> used by the economists in assembling these estimates, the <u>data</u> which they used were necessarily meager and some of them were 10 to 15 years old. There has been no direct attempt to design field experiments to find the economic costs of aldrin-dieldrin replacement. In the case

of crops other than corn, there was an even graver deficiency of data upon the economics of insecticide use.

These rough economic analyses provide an incomplete picture. The reduction in corn yield by aldrin replacement could be as little as 1% nationally. Should one neglect this in view of the current national corn surplus? Should one be concerned if aldrin-dieldrin withdrawal prohibited the continuance of certain crops in particular farm areas? Or if it severly affected (or even eliminated) crops such as mangoes, pineapples or sweet potatoes, whose national economic impact is relatively slight, but whose local importance may be major? Our response to such questions was to exercise caution in recommending withdrawal.

Another factor is the steady growth of insect resistance for aldrin-dieldrin, especially in soil insects. We believe that this factor will lead to a progressive decrease, especially in aldrin use, and that this will cause an important reduction in dietary intakes; corn is the major usage of these compounds, and most dietary intake is from meat and dairy products (which derive much of their nutrition from corn) so that human intakes are likely to be geared to usage on corn.

An unexpected problem is that farmers may not know what pests they are controlling! Thus we received 16 petitions from very many rice farmers telling us how important was their use of dieldrin for seed treatment to control rice water weevil. But our USDA experts told us that this use of dieldrin was now ineffective for

rice water weevil, but important for the grape colaspis. Even for soil use on corn there was disagreement about just what pests are controlled. All that the farmers can know is that his regular use of such compounds gives protection from undue insect damage. He is not likely to omit treatment in order to find out what pests are truly damaging that year.

The problem is compounded by the fact that corn farming (for instance) has been improved to give very much larger yields, up to two-fold increases in the last 15 years. This intensive farming uses heavy investments in equipment, fertilizer and herbicides as well as insecticides, leading to a high investment enterprise requiring very efficient growth conditions. The economics are correspondingly more fragile.

Other committee reviews.

We are keenly aware of the fact that we are a part-time committee whose recommendations are based upon a reading of the literature but only a few meetings together. But our conclusions, independently arrived at, are consonant with those of the other expert committees who have examined the evidence in recent years. In 1965, the Popper Committee (advisory to FDA) bearing in mind principally the possible dangers to humans (but not to wildlife) recommended continued use of aldrin and dieldrin for certain pesticidal purposes. Also in 1965 the Jensen Committee (of NAS-NRC) pointed to the importance of use of pesticides, including persistent ones. In 1967 the Gunther Committee (advisory to FDA)

reviewed aldrin and dieldrin use, and concluded that current tolerances were acceptable, but pointed to the need for additional research e.g. on carcinogenicity in more species, and epidemiological studies. In 1969 a committee reporting to the Ag. Res. Service, USDA, proposed that persistent pesticides as currently used were necessary, but called for "steps to be taken to reduce the needless or inadvertent release" of them. It also called for further research e.g. on additional means of control and on long term effects on man and wildlife. In 1969 the Wilson Committee (advisory to the British Dept. of Education and Science) found that there was no evidence that dieldrin was harmful to man, and so "no high priority can be presently assigned to the removal --- of dieldrin"; but that it is "undesirable that the human environment should contain substances capable of producing toxic effects". In 1969 the Mrak Commission (advisory to the Secretary of HEW) recommended that aldrin and dieldrin be restricted "to specific essential uses which create no known hazards to human health or to the quality of the environment and which are unanimously approved by the Secretaries of Health, Education and Welfare, Agriculture and Interior". The Commission noted that it was "impractical to attempt to eliminate the residues of such pesticides from foods by the application of zero tolerance techniques" because analytical techniques are so extraordinarily sensitive. In 1970 the Joint FAO/WHO expert committee recommended a series of tolerances of aldrin or dieldrin in foods such that the daily intake in man should

not exceed 0.1 ug/kg/day (roughly twice the typical U.S. intake).

Conclusions.

We find no evidence of human injury from present or past use of aldrin or dieldrin. Nevertheless the facts that fairly low levels of dieldrin can cause cancer in mice and interfere with reproduction in some birds are matters for concern, and point to the need for more careful evaluation of the hazard to man. There is clear evidence that past usages have been deleterious to wildlife. Several such past usages have been voluntarily abandoned by Shell Co. Nevertheless, we feel that we must strive to find alternate methods of pest control, including nonchemical methods, for all compounds which lead to persistent residues in humans or wildlife, even when such residues are not demonstrably harmful. How can we move towards this objective?

When aldrin or dieldrin can be safely and economically replaced by nonpersistent pesticides they should be so replaced. Several practices which can readily lead to damaging effects upon non-target organisms should be abandoned now in spite of the difficulty of economic replacement, including all applications which lead to contamination of aqueous environments such as rice fields and waterways

The direct application of aldrin or dieldrin to soils leads to negligible leaching or other transfer from those soils, and environmental contamination is thus very small except where substantial erosion takes place. One of the few studies to estimate

the amount which volatilized indicates that 3% escapes this way, and thus contaminates the environment directly (we would like to see more extensive data upon this point).

Recommendations.

The following recommendations are designed to build a basis of facts on which permanent recommendations can be formulated, and to eliminate now those uses of aldrin or dieldrin which result in significant environmental contamination (especially to waterways). We believe that applications directly to soil or to materials buried in soil (e.g. termite control in foundations, and seed treatments when properly applied) lead to little subsequent movement of these insecticides, and should be permitted.

In the following recommendations, we use the term "experts" and "acknowledged authorities" advisedly. The EPA must seek contractual or other arrangements with individuals and institutions accepted as authorities by their peers in the country at large.

1. A committee of experts in chemical carcinogenesis should be formed to propose specific experiments and to agree upon suitable protocols to provide a firm indication of the extent of carcinogenic hazard. These experiments should include studies (in at least two vertebrate species) on the effects on the progeny of mother fed dieldrin during pregnancy and nursing, the progeny also being fed dieldrin thereafter.

2. The economic consequences of total withdrawal of aldrin and dieldrin should be explored in depth: On all major crops,

actual experimental studies must be performed to obtain new, reliable data provided by acknowledged authorities, and should include studies with and without alternative nonpersistent pesticides, over a series of years, and in appropriately distributed geographical areas.

3. The fraction of aldrin and dieldrin which escapes by volatilization following application to a variety of soils, under conditions of application and treatment levels commonly used in pest control, should be measured by acknowledged authorities.

4. Monitoring stations should be established in the U.S. and abroad, at which air and water samples can be taken at fixed places over a series of years, and analyzed by unambiguous procedures for aldrin and dieldrin. The intent is to study whether the restrictions we propose do indeed lead to a progressive removal of these compounds from the environment. Agreement should also be sought amongst a group of experts for unambiguous procedures for determination of aldrin and dieldrin in extracts of air, soil, water, food and human and nonhuman tissues. Such procedures should be standardized in the U.S. and preferably internationally as well.

5. The following uses of aldrin or dieldrin should be disallowed.

(a) All applications by aircraft.

(b) All foliar spraying or dusting.

(c) Moth proofing by the hot acid dye bath method or related methods in which residues are discharged into waterways or

settling ponds.

(d) All uses, whether by homeowners or pest-control operators, in homes, barns, poultry operations or other structures occupied by humans or livestock.

(e) Use upon turf (including lawns and non-grazing grassed areas) except as supervised or controlled by trained or licensed pest-control operators, greenskeepers and nurserymen.

(f) Any use which involves application to streams, ponds, lakes, flooded areas or any other aquatic environments.

6. Specific uses of aldrin and dieldrin which we believe to be valuable and not harmful include:

(a) Direct applications to soils.

(b) Seed treatments, when the treated seed is labelled "not for food use".

(c) Dipping of plant roots or tops during transplantation.

(d) Treatment of foundations, by current procedures, for termite control.

(e) Use of treated hot-caps.

7. Because our recommendations are based upon evidence which, although the best available, is still not complete; we recommend that the environmental and economic effects of the proposed restrictions be reviewed 5 years after their imposition. By that time, the completed results of recommendations 1,2,3 and 4 should be available.

APPENDIX I

USES OF ALDRIN AND DIELDRIN

Mahlon L. Fairchild and R. L. Doutt

The amounts of aldrin and dieldrin used in the United States substantially decreased from 1966 to 1970 (from ca. 19 million pounds aldrin, one million pounds dieldrin in 1966 to ca. nine million pounds aldrin, 0.6 million pounds dieldrin in 1970)(232). Among the reasons for this reduction are (a) an increased resistance to aldrin/dieldrin by some target pests, (b) the development and availability of substitute materials, and (c) the desire to restrict the use of persistent organochlorine insecticides to essential needs.

A few examples of increased resistance to aldrin/dieldrin are as follows:

(a) The rice water weevil, Lissorhoptrus oryzophilus, now resistant to aldrin treated rice seed(159).

(b) The tobacco wireworm, Conoderus vespertinus, and the southern potato wireworm, C. falli, in North Carolina(186).

(c) The seed-corn maggot, Hylemya platura, in Illinois(107) and New York(141).

(d) The pear psylla, Psylla pyricola, in Washington(177).

(e) The European chafer, Amphimallon majalis, in New York(247).

There are potential substitutes for aldrin/dieldrin in many situations. Carbofuran is promising in the control of two major pests of berry crops and ornamentals in the Pacific Northwest,

namely the strawberry root weevil, Brachyrhinus ovatus, and the black vine weevil, B. sulcatus(230). Carbofuran is also effective against the nut curculio, Conotrachelus hicoriae(36). Although dieldrin reduced cat facing in peaches caused by tarnished plant bugs, Lygus lineolaris, and stink bugs, Euschistus, so did Gardona and endosulfan(20). Where resistance had not developed, dosage rates for dieldrin used against scarabeid grubs in turf were sucessfully reduced from 3 lbs./acre to less than 1 lb./acre(244) and it is suggested that where applications are carefully timed such products as diazinon, Dursban and Carbaryl are available substitutes(247). It is established that the same amount of aldrin is less effective against arthropods in soils of high organic content than in sandy loam whereas possible alternative products are either not affected by such organic matter (phorate) or only slightly affected (parathion, Dyfonate®)(38). The screening of materials for control of maize bill bug, Sphenophorus maidis, showed that there are insecticides other than aldrin which can be used as a preplant, broadcast, disked-in treatment(59).

Wireworm control studies on potatoes in Idaho with side-dressed and broadcast insecticide applications showed Bay-38156 and disulfoton side-dressed at rates of 8 and 18 ozs. A.I./1000 ft. row, Agritox® and Dasanit® side-dressed at .8 and 1.8 oz. A.I./1000 ft. of row and propoxur was side-dressed at .9 and 1.7 oz. A.I./1000 ft. of row resulted in significant reductions in wireworm injury.

Dyfonate and Agritox controlled wireworms as well as chlorinated hydrocarbonous materials, represented by chlordane(221).

American Cyanamid 18133 at 1.2 lbs. A.I./acre, Heptachlor at 1.5 lbs. A.I./acre and Hercules 5727 at 1.13 lbs. A.I./acre, seemed to give significant reduction in sand wireworm injury to corn in Missouri(143).

Bayer 25141, diazinon, GS-13005, Kepone, N-290, Niagara 9203 and parathion gave good results for wireworm control on cigar-wrapper tobacco(241).

Investigations of controls on wireworms on potatoes in eastern Washington found that Telone®, D-D® mixture, ethylene dibromide, diazinon, parathion, and phorate, gave economic control of the Pacific Coast wireworm on potatoes in sandy loam soil(198).

Harris (1965) reported diazinon baits, Kepone baits, parathion granules and diazinon granules to be somewhat effective against wireworms in sweet corn. Brett et al.(1966) reported that diazinon gave poor control of wireworms in sweet potatoes when it was incorporated into the soil prior to planting, but a foliage-surface application during August gave good control. Diazinon gave good control when applied on the surface late in July at the rate of 3 lbs. active ingredient per acre in a granular formulation.

N-2790 and Zenophos at 5 lbs./acre broadcast and worked into the soil shortly before planting, gave more than 90% wireworm-damage-free potatoes compared with 26 to 55% damage-free potatoes in untreated soils. Diazinon at 7 lbs./acre and Bay 25141, and Bay

37289 and UC 10854 at 10 lbs./acre also gave more than 90% damage-free potatoes(31).

Fumigation of the soil in autumn with ethylene dibromide, Telone® or dichloropropane-dichloropropene mixture has given excellent control of Pacific Coast wireworms. Dyfonate at 2 lbs./acre, 3/4 lb./acre of diazinon or parathion, and 4 lbs./acre of carbofuran or Bay 37289 also gave excellent control when broadcast(196). Onsager and Foiles(197) reported when granular carbofuran, Dyfonate, and parathion were applied in mid-March for control of the Great Basin wireworm, and the Pacific Coast wireworms on summer potatoes that side-dress treatments at a rate of about 2.3 lbs. of toxicant per acre gave better average control with less variation than broadcast treatments with 4-8 lbs. of the same toxicant per acre.

Flooding infested fields for 6 to 7 days with an inch or two of water during extremely hot weather has been reported to kill nearly all wireworms. Soil temperature must remain above 68°F for the entire period for best results. In contrast, when the upper 15 inches of infested soil is allowed to become very dry for several weeks in summer, most of the wireworms, especially the younger ones, are killed. Drying has been recommended to keep wireworms below economic levels on irrigated lands.

Plowing fields in the summer during the pupal stage has been reported to reduce wireworm numbers. Mechanical injury to the worms and exposure to summer heat and low humidities account for most of the mortality at this stage.

Crop rotation in certain areas has been reported to reduce wireworms. The growing of truck crops in the same area continuously will usually cause a build-up of wireworms. The growing of potatoes in a short rotation with clover or grain is undesirable because of wireworm build-up.

In Georgia in peanut fields Abate®, monocrotophos, Dursban®, Dyfonate, trichlorfon, methomyl, and Monitor® gave higher than 90% control of granulated cutworm larvae within 24 hours after application(138).

DDT, Bay 37289, Dursban, and trichlorfon have been evaluated for control of the dark-sided cutworms in rye grown in rotation with tabacco. In plots using artificial infestations DDT and Dursban gave 100% control while Bay 37289 provided 90% control, trichlorfon at 1 lb. per acre was not sufficiently effective. It was noted that DDT, Dursban and Bay 37289 provided 88-90% control of heavy natural infestations. It was noted, however, that the rapid dissipation of the residues of Dursban and Bay 37289 would require a split application to give adequate control(116).

Studies indicated that Dursban, Bayer 37289, and parathion warrant further investigation as possible alternatives for the cyclodiene insecticides as soil treatments for control of the black cutworm. It was noted, however, these materials should be applied when the larvae are in the early instars to be effective. Soil type, moisture, method of application, and formulation must be taken into consideration for effective control(113).

Further studies showed three compounds having potential as

possible replacement for cyclodiene compounds as control agents for dark-sided cutworms. These compounds were N-2596, phoxim and chlorphoxim. Chlorphoxim seemed to be somewhat less effective than the other two compounds(115).

Unpublished studies (227) indicated that Niagara 10242 (Furadan®, a combination of thimet and A.C. 47470, Dursban, Sevin®, and G.S.-10133 as well as Baygon® showed promise as effective insecticides against black cutworms.

Diazinon, Dyfonate, dylox, Sevin, Bay 37289, GC-6506, EI-52160, and Thimet + EI-4740 have been shown to be effective against cutworms (224).

Begg et al. in 1963(15) reported that soil treatments in general did not reduce the infestations of the black cutworm to non-economic levels during the first seven days after treatment. It apparently took some time for the larvae to contact a lethal amount of an insecticide in the soil. The results of field test conducted with eight insecticides indicated their relative effectiveness in test could be rated endrin>aldrin = dieldrin = heptachlor > DDT = Dylox>Guthion = Sevin.

According to work done by Harris and Mazurek in 1961(110), the regression line slopes would indicate that a small increase in the rate of application of dieldrin should result in more effective control of cutworms. Conversely, large increases in rate of applications of Dylox, Guthion and Sevin would result in little or no increase in insecticide efficiency.

Laboratory studies(109) conducted to determine the toxicity of 3 insecticides, DDT, Dursban, and Galecron®, to the various stages in the life cycle of the darksided cutworm indicate that DDT was effective against the early larval stages, but ineffective against the later larval stages, eggs, pupae, and adults. Dursban was effective against eggs, early larval stages, and adults, but ineffective against the later larval stages and pupae. Galecron was highly selective and was effective against eggs.

Laboratory tests(114) indicated that 5 of 7 insecticides tested as direct-contact poisons against 3rd- and 4th-instar variegated cutworms were more effective than aldrin. The most toxic was Lannate®> DDT> parathion > Dursban> Ciba 8874. Birlane® and Bayer 37289 were slightly less toxic than aldrin. As soil surface applications, Dursban and aldrin were highly effective, DDT, parathion and Bayer 37289 were only slightly less so, and Lannate was ineffective. Soil-surface applications of Dursban were 2-4 times as effective as soil incorporations. Dursban, DDT, and Lannate were all highly effective as stomach poisons. Lannate was 32 times more effective as a stomach poison than as a soil insecticide.

Greenhouse studies have indicated several baits to be effective against black cutworm populations. The baits were: Trichlorfon Bait C, CL-47470 - 2% apple pomace, Abate - 2% apple pomace, ethyl parathion - 2% apple pomace, mirex - 1.25% bran, TDE - 5% bran, and carbaryl - 5% bran(225).

Studies by Harris in 1962 indicated that there was very little

resistance building in black cutworms in certain areas of Canada to soil insecticides. These studies also indicated that in general organophosphates were less effective against black cutworms than cyclodiene insecticides.

Several methods have been suggested for controlling white grubs. Hot water has been suggested to control the Japanese beetle in the soil, on the roots of nursery plants(73). Researchers have reported on the success of using traps baited with geraniol and eugenol for control of white grubs, particularly the Japanese beetle(79,182).

The effectiveness of lead arsenate to control white grubs has been explored by researchers(74,78). Napthalene and Derris have been reported to be helpful repellents of the Japanese beetle.

Many studies have been conducted using cyclodienes to control grubs. Cyclodiene compounds such as TDE, chlordane, methoxychlor as well as ethylene oxide, methyl bromide, lead arsenate and several imported parasites have been reported to control the larvae of the Japanese beetle (Fleming, 1937), (101). Paris green and its homologues have been effective insecticides against Japanese beetle larvae(77).

Burrage and Gyrisco(29,30) studied the control of the European chafer in pasture sod. The test conducted by Burrage and Gyrisco included six materials - parathion, dieldrin, aldrin, chlordane, BHC, and DDT. Parathion and BHC did not perform as well as the other materials. Grambell(91,92,93,94,95,96,97,98) did

extensive work on controlling the European chafer. Most of these works included the use of cyclodiene insecticides for control. Tashiro(243) reported that methyl bromide fumigation gave satisfactory control of the European chafer in nursery stock.

Carbon-disulphide emulsion as well as lead arsenate could be used to control Asiatic beetle larvae in lawns(102). Several parasites have been reported to attack Scarabaeidae larvae(Fleming 1936);(32,60,83,86,148). Milky diseases in beetles have to be reported to give rather effective control of several white grubs(119) (Fleming, 1933).

There is very little information about the hazards to man and the environment involved in the use of such substitute materials. There is no evidence that aldrin/dieldrin are generally more harmful to beneficial insects than some of the possible substitute materials. It is known that aldrin/dieldrin are highly toxic to bees(3), but so are carbamates and organophosphates. It appears that aldrin/dieldrin are no worse than substitute materials in tests against certain predators and parasitoids and dieldrin was reported to be somewhat less toxic to syrphids than certain other insecticides including carbaryl, endosulfan and parathion(220).

The bluegrass bill bug, Sphenophorus parvulus, became a pest on lawns previously treated with dieldrin(246) (and other cyclodiene insecticides), but there are in general very few reports of outbreaks of other pests following the use of aldrin/dieldrin. Although increases in mites and aphids have been mentioned, really adequate documentation is lacking(13).

The use of aldrin/dieldrin for control of the subterranean termites Reticulitermes spp., poses minimal and insignificant hazards to man or his environment and offers the most effective and durable control of these structural pests at this point in time. The applications are generally made as water emulsions to the soil under foundations and footings of buildings. Here there is a minimum of weathering, erosion, or other disturbance and the persistent characteristic is a tremendous asset giving control for 20 years (188). Furthermore, tests have shown that dieldrin can be used in the mix water for concrete to prevent tubing over by Reticulitermes (14). Aldrin and dieldrin are somewhat less effective against the Formosan termite, Coptotermes formosanus.

Similarly, there are minimal environmental hazards involved in the use of aldrin/dieldrin against other structural, wood-boring pests, or with dieldrin in stump treatments to control pales weevil, Hylobius pales, or northern pine weevil, Pissodes approximatus(19). In this latter case, however, endosulfan is an effective substitute.

The use of dieldrin as a seed protectant with its application restricted solely to planting seed of certain specified crops is also without demonstrable hazard to man or the environment. The use of such treated seed eliminates the need for soil applications in some situations(117). There is no documentation of any appreciable hazard in the present use of dieldrin as a primary seed protectant on cotton, corn (sweet, field, pop), beans (green, snap, lima and

black-eyed-peas), peas, cowpeas, cucumbers, summer squash, onions, small grains (oats, rye, barley, wheat, grain sorghum), and soybeans. The only hazard involved is that such planting seed might be used inadvertently for food or feed.

The use of dieldrin for turf insects and for insects on commercially grown ornamentals and in nurseries needs to be restricted to the supervision and control of trained or licensed pest control operators, greens keepers and nurserymen. This is proposed by one registrant, Shell Chemical Co. Labels should restrict the sale to commercial users, and dosages should be minimal and may need to be revised downward in view of the control of Scarabaeid grubs with one-third the ordinary dosage(244). These uses of dieldrin need constant surveillance and revision in view of the developing resistance in the pests and the increasing availability of suitable substitutes.

There are no scientific data on actual losses caused by Fuller's rose beetle to citrus. It does not rank as a major pest in California citrus, but apparently is considered damaging in Florida(232). A substantial amount of aldrin has been applied against this insect (estimated 143,300 pounds in 1970). The magnitude of this application strongly suggests that the damage actually caused by Fuller's rose beetle needs a thorough scientific appraisal. Until such information is obtained, there is no scientific basis for advisory comment. This lack of information on pest population tolerance levels (sometimes called economic threshold) is not limited to the Fuller's rose beetle, but applies to most agricultural pest problems.

Conclusions

1. Although pesticides such as aldrin/dieldrin should be applied solely on the basis of established need, there is almost a complete lack of reliable scientific data to establish this basis for treatment of most agricultural pests. Tolerance levels for such pests need to be investigated, and better information on the precise time, place and manner of application needs to be obtained.

2. The use of aldrin/dieldrin for control of subterranean termites gives effective protection to wooden structures and does not pose any demonstrable hazard to the environment.

3. The hazards in the use of dieldrin as a seed protectant are minimal when it is restricted solely to the planting seeds of the following crops: cotton, corn, beans (green, snap, lima and black-eyed peas), peas, cowpeas, cucumbers, summer squash, onions, small grains (oats, rye, barley, wheat, grain sorghum), and soybeans.

4. Certain turf insects are becoming tolerant of aldrin/dieldrin and substitute materials are available. Where Scarabaeid grubs are not resistant, the dosage rates can be reduced.

5. The application of aldrin/dieldrin for turf insects and for insects on commercially grown ornamentals and in nurseries should be restricted to trained or licensed pest control operators.

APPENDIX II

TOXICOLOGY, METABOLISM AND BIOCHEMICAL EFFECTS ON NON-HUMAN SPECIES

Florence K. Kinoshita

Toxicology

A. Mammals: The acute oral toxicity of aldrin or dieldrin has been summarized by Hodge et al.(129) as being between 20 and 70 mg/kg for 12 different mammalian species. Of these species the cat was the most sensitive; the cow, rat, guinea pig, mouse and monkey were intermediate in susceptibility: the dog, pig and sheep were the most resistant. The young of a species may be more susceptible to the toxic effects of dieldrin than the adults(129). A sex difference in toxicity has been noted in the rat. Female rats are less resistant to dieldrin toxicity [LD_{50} females = 51 mg/kg; males = 64 mg/kg](120). Male rats, however, have been reported to be more susceptible to the toxic effects of aldrin orally [LD_{50} males = 39 mg/kg; females = 60 mg/kg](82).

The symptoms of poisoning after an acute oral dose appear within 30 to 60 minutes after administration(120). Death occurs within 1 week. After the intravenous administration of a toxic dose [LD_{50} to female rats = 8.9 mg/kg] the symptoms appear within 2 to 5 minutes and death occurs within 40 minutes. The symptoms that occur are related to effects on the central nervous system. These include hyperexcitability, hyperactivity, incoordination, exaggerated body movements which later lead to convulsions and depression of the central nervous system and finally to death(9).

The mechanism of toxic action of aldrin and dieldrin is not

known, although it is known that the nervous system is involved. Aldrin has been shown to cause stimulation of the parasympathetic nervous system peripherally leading to salivation and slowing of the heart(89,90). Dieldrin has no peripheral action on the parasympathetic system. Both aldrin and dieldrin have central effects which lead to parasympathetic stimulation(88,89). There has been a report of the effects of aldrin and dieldrin on brain betaines(131). The most recent proposal for the mechanism of action of aldrin and dieldrin has been that of Shankland(229) who reports evidence of dieldrin affecting the cholinergic system of insects by its effect on presynaptic acetylcholine release. This mechanism has as yet not been verified in vertebrates.

The effects of a single oral dose of dieldrin are long lasting; the effects may persist for 3 weeks. If an animal is given a second dose of dieldrin during this time, there is greater toxicity in terms of mortality than would be expected(9,120).

With short-term administration of repeated doses there is no mortality with doses of less than 25ppm in the diet(129). In chronic studies monkeys fed 5 ppm dieldrin died after 1 year(129). In other mammalian species mortalities are caused by levels of 10 to 20 ppm. It has been noted by several investigators that starved rats or rats on restricted protein diets are more susceptible to the toxic effects of dieldrin administered as part of the diet(50, 136, 238).

Histopathology after the administration of aldrin or dieldrin for long periods of time indicates changes in the liver, kidneys, lungs, brain and vasculature(104,144).

An increase in the liver weight to body weight ratios is seen in rats fed 2 ppm aldrin or 0.5 ppm dieldrin and in dogs fed 3 ppm aldrin or dieldrin(129). The increase in liver weight is accompanied by a hypertrophy of the smooth endoplasmic reticulum of the liver(134). A fatty metamorphosis in the central zone with increased amounts of basophilic material and hepatic cell necrosis predominantly near the central zone, but also scattered throughout the parenchyma occurs in the mouse fed 3 ppm of aldrin or dieldrin(144). Higher levels of dieldrin (25 to 50 ppm) cause enlargement of liver cells around the central vein and margination(147).

With levels of 3 ppm aldrin given to mice moderate congestion of the blood vessels of the lungs, mild alveolar emphysema and minute hemorrhages in the alveoli have been reported(144). These authors also report the presence of bronchiolar carcinoma and benign pulmonary adenomas in mice fed 5 ppm aldrin.

The kidneys of mice fed 3 ppm aldrin or dieldrin and higher levels have been reported to have vascular congestion, focal glomerulonephritis, swelling of Bowman's capsules and dilation of the convulated tubules and cloudy swelling(144).

One group of investigators(104) report that rats being fed 2.5 to 10 ppm dieldrin had occasional neural spasms. The nervous tissue of these animals demonstrated vascular congestion in the meninges, edema in the parenchyma and swollen upper motor neurons. Also there was focal degeneration and necrobiosis in the cortical laminae. Cerebellar lesions included cloudy swelling, Purkinje's cell degeneration and degeneration foci in the granular layer. Brainstem

lesions included neuron and myelin degeneration, glial swelling and adventitial proliferation. The craniums of these rats contained increased amounts of clear cerebrospinal fluid. With the feeding of 0.31 to 10 ppm dieldrin edema of the leptomeninges was seen. The brain concentrations of dieldrin in those rats showing convulsions was between 9 and 11 ppm. The vascular effects of dieldrin in rats fed 0.31 to 1.25 ppm were reported to be of a degenerative nature, while those in rats fed 2.5 to 40 ppm were inflammatory in the males and necrotic in the females(104). These observations have not been confirmed by other reports.

The effects of dieldrin on reproduction have been studied in rats, mice and dogs. At levels of 0.31, 1.25, 2.5 and 10 ppm dieldrin, there is a decrease in the weaned litter size of rats (87,103). At the higher levels of dieldrin intake by the dam, the pups died in convulsions. Death often occurred within the first 5 days in pups due to the lack of nursing. In mice 3 and 5 ppm aldrin lead to decreased viability in the 2nd generation and lower fertility in the 3rd generation. 10 ppm dieldrin decreases fertility in mice, while 3 ppm dieldrin decreases lactation in the 2nd and 3rd generations(144). The summary by Hodge et al.(129) indicates that the lowest level of aldrin having an effect on reproduction in rats is 12.5 ppm and the lowest level of dieldrin having an effect is 2.5 ppm. The later study(103) indicates, however, that reproductive effects are seen with 0.31 ppm dieldrin; these data are not confirmed. In dogs the lowest level of aldrin and dieldrin having an effect on reproduction are 8 and 25 ppm respectively(129).

B. Birds: The acute oral toxicity of aldrin has been reported for four species(252). The LD_{50}'s for these species are 6.6 mg/kg in the female bobwhite quail, 16.8 mg/kg in the female pheasant, 29.2 mg/kg in the male fulvous tree duck and 520 mg/kg in the female mallard duck. The lowest daily dose that can be tolerated for 30 days by the mallard is 5 mg/kg.

The symptoms of poisoning by aldrin in birds include ataxia, circling, low carriage, closure of the nictitating membrane, tremors, phonation, wing-beat convulsions, seizures and opisthotonos. Death occurs from 1/2 hour to 10 days after treatment.

Feeding studies in birds with aldrin indicate a no-effect level of about 1 ppm(56). One day old quail fed feed containing 1 ppm aldrin survived for 47 days, one day old pheasants started on a diet containing 5 ppm aldrin exhibited 100% mortality by the 46th day. Symptoms of poisoning at these levels occur 48 to 72 hours after the initiation of treatment; the symptoms are those that are seen in adult birds with acute poisoning. Five ppm aldrin will cause 100% mortality in adult quail and pheasants.

The effects of aldrin on reproduction in birds indicate a decrease in egg production with a level of 1 ppm, with a cessation of egg production by the 6th week(56). The hatchability of the eggs laid by birds fed 10 ppm decreased as did the fertility. There was no effect on chick viability at this level.

The acute oral toxicity of dieldrin has been determined in

various domestic and wild species of birds. In the chicken the oral LD_{50} for adults has been reported to be between 20 and 30 mg/kg while other studies indicate that 44 mg/kg causes no mortality(129). In wild species the LD_{50} is reported as being 381, 79, 23, 70, 27, 48, and 9 mg/kg for the mallard, pheasant, chukar, coturnix, pigeon, sparrow and gray partridge respectively. The acute dose for the Canada goose is between 50 and 150 mg/kg. The daily dose that can be tolerated for 30 days is 2.5, 1.25 and 5.0 mg/kg for the fulvous tree duck, gray partridge and mallard respectively(252,253). The symptoms of acute poisoning are hyperexcitability, jerky gait, ataxia, dyspnea, myasthenia, fluffed feathers, immobility, opisthotonos and terminal wing-beat convulsions. Death occurs within 1 to 9 days after poisoning.

In feeding studies the administration of 5 ppm dieldrin to day old quail causes 100% mortality, while 0.5 ppm has no effect on survival. One ppm causes 100% mortality after 76 days. A level of 5 ppm will cause 100% mortality in pheasants by the 68th day(55). The susceptibility of adult birds to repeated feeding of dieldrin is not as great. Adult pheasants fed 100 ppm dieldrin exhibit 100% mortality between 10 days in the males and 39 days in the females. In quail 10 ppm dieldrin has no effect while a level of 20 ppm causes 50% mortality between 13 and 63 days.

There have been reports that 10 ppm dieldrin fed to quail causes a decrease in the hatchability of eggs and the survival of chicks(55,56), while other reports indicate that levels of 20 and 30 ppm are needed

to cause a decrease in egg laying(257). At the 20 and 30 ppm levels there is increased chick mortality by the 3rd day after hatching(257). In pheasants there is a slight decrease in egg laying by birds fed 25 ppm, while 50 ppm significantly decreases egg laying(84). The survival of the chicks from the eggs of female pheasants fed 50 ppm is decreased by 35%. In the gray partridge 3 ppm dieldrin given as a pellet did not affect fertility or egg hatchability, however, there was a slight increase in mortality in the shell(193). The growth rate and chick survival after hatching were not affected by this level. Dieldrin at levels of 1.6, 4 and 10 ppm given to penned mallards caused a decrease in eggshell thickness(162).

The population of wild birds in areas treated with dieldrin did not change after dieldrin application. The clutch size and hatchability of gallinules are not affected when eggs contain as much as 13 ppm dieldrin(40). The use of rice bran contaminated with residue levels of dieldrin to feed leghorn hens had no effect on egg production, hatchability or chick survival(80,100). There is a correlation between the amount of dieldrin found in the eggs and the amount of dieldrin fed to the birds. A dietary level of 20 ppm fed to quail can cause over 45 ppm to be found in the eggs after 7 weeks(257).

C. Fish: There are few actual experimental data available on the toxicity of aldrin or dieldrin to fish. The median tolerance limits (TLm) which is equivalent to the LD_{50} for a specified exposure period has been reported to be 0.0155, 0.012, 0.0075 and 0.067 ppm for periods of 24, 48, 72 and 96 hours respectively for the pumpkinseed

sunfish(35). In a 96 hour exposure period levels of 0.32, 0.0155, 0.0087, and 0.0075 ppm were highly toxic while 0.0056 ppm caused no mortality. However, the level of 0.0056 ppm caused 100% mortality by the end of 1 week. The 96 hour TLm for minnows, blue gills, goldfish and guppies is between 0.015 and 0.037 ppm(129). Exposure of steelhead trout to 1.2 ppb for 45 days leads to 100% mortality(41).

The levels of 0.0056 and 0.0032 ppm dieldrin caused toxic effects in the pumpkinseed sunfish by decreasing cruising speed and increasing the consumption of dissolved oxygen. Difficulty in orientating to the current and an increased sensitivity to sunlight were also noted at these levels(35). A concentration of 0.00168 ppm dieldrin while causing no mortality causes an increase in oxygen consumption and a decrease in cruising speed in sunfish.

Exposure of guppies to a level of 0.01 ppm dieldrin leads to the production of no fry after the 32nd week of exposure(34). These authors noted an initial increase in population which they attributed to a change in the behavior of the adults resulting in less predation of the young by the adults. Exposure of steelhead trout to a level of 0.39 ppb dieldrin results in only a 3% survival rate of fry to age 130 days(41). The growth of trout is not affected by levels of 0.12 ppb and below.

D. Invertebrates: Experimental data on invertebrates are in shorter supply than that for fish. The 5 day median tolerance limits for aldrin for the Louisiana red crawfish weighing 0.5 grams is 56 ppb(124). These authors report that up to 200 times this amount in

the soil has no effect on the survival or growth of the crawfish.

Oysters exposed for 10 hours to water containing 1 ppm dieldrin exhibit physiological irritation which is seen as a continual opening and closing of the valves(33). This continual opening and closing of the valves indicates an abnormal feeding process. After 2 weeks of exposure to 0.1 ppm dieldrin the oysters are only half as active as the controls. There is interference with shell deposition by the oyster in the presence of dieldrin. The oyster will also store chlorinated hydrocarbons when they are present in concentrations of 0.1 ppb or more.

Pharmacodynamics-Metabolism

After the oral administration of dieldrin it is absorbed from the upper gastrointestinal tract and is found in the liver, kidneys, mesenteric and retroperitoneal fat after 1 hour. Redistribution occurs and there are increasing amounts found in the fat and genital organs while the concentration remains constant in the liver and kidneys. At the end of 2 days after a single dose the highest concentrations are in the mesenteric and retroperitoneal fat(120).

Biliary excretion occurs(136,151). After a single dose about 90% is excreted in the feces while the urine contains about 10%(45,120). If the animal is starved or on a restricted diet, excretion is accelerated(120).

With continuous administration in the food or daily by the oral route dieldrin is stored in the fat(10). The concentration in the tissues is a function of the daily intake level(26). An upper limit of

tissue concentration is reached which is characteristic of the intake level. A balance is reached between intake and elimination and storage. The length of time required to reach a plateau storage level is also a reflection of the daily intake level(53, 142). The concentration of dieldrin in blood is a reflection of the concentration in various tissues for man, rats, dogs and birds(211). The storage ratios in the tissues of mammals is blood:liver:fat(53) and in birds it is fat:liver:muscle:brain(210). In birds (chickens) the amount of dieldrin in blood is also a reflection of the amount that is in eggs(210). In the rat a sex difference in storage is seen. Female rats store more dieldrin than do males(151) and the excretion of labeled material is greater by the male rats than by the females(45). Once intake is terminated the amount of dieldrin in the tissues decreases(210). The biologic half-life of dieldrin in the fat of rats is approximately 4.5 days(10).

In pregnant mammals dieldrin can cross the placental barrier and concentrate in the tissues of the fetus(6,103). There is also excretion of dieldrin in the milk of the lactating animals so that a nursing pup has the same distribution in tissues as the adult by the end of 2 days. Dieldrin is also excreted in the milk of lactating dairy cattle(22).

The administration of aldrin to animals and insects leads to the formation of the epoxide, dieldrin(26,156,234). In the rat this metabolic conversion occurs in the liver microsomes and is inducible (26.239). The conversion of aldrin to dieldrin is also faster in the

male rat than in the female rat(268). The epoxide is further metabolized to hydrophilic compounds which are excreted in the urine and feces(234). At least 6 metabolites have been isolated. One of the metabolites found in both urine and feces and possibly existing as a glucuronide conjugate(68,120,156,157) has been found in rabbits, rats and sheep. This is the compound aldrin trans-diol.

The trans-diol is less toxic than either aldrin or dieldrin by the intravenous and oral routes. The oral LD_{50} is reported to be 1250 mg/kg to the mouse compared with 75 mg/kg for dieldrin and 45 mg/kg for aldrin(156).

Photoisomer: Upon exposure to sunlight a photoisomer of dieldrin is formed. This compound is found as a residue on some agricultural commodities(260). Acutely this photoisomer is more toxic than dieldrin to rats, mice, guinea pigs and pigeons. The acute oral LD_{50} for these species is 9.6, 6.8, 2.3 and 90 mg/kg respectively compared to 47, 77, 18 and 250 mg/kg for dieldrin in a study done by a single group(27). To hens dieldrin is more toxic than the photoisomer, while in the beagle there is no difference.

It is reported that both photoaldrin and photodieldrin are more toxic to freshwater animals than either aldrin or dieldrin(229). The 24 hour LD_{50} to the bluegill for aldrin, photoaldrin, dieldrin and photodieldrin are 0.26, 0.09, 0.17 and 0.03 ppm respectively. The 24 hour LD_{50} to the minnow for dieldrin and photodieldrin are 0.024 and 0.010 ppm respectively. The LT_{50} (time to kill 50% of the population at a given dose) is shorter for photodieldrin than

for dieldrin for crayfish, planaria, tadpoles, guppies, bluegills and minnows.

Subacutely, 3 to 10 ppm of the photoisomer is lethal to the mouse, while 30 ppm of dieldrin is required for mortality(260).

Biochemical Effects

One of the effects of aldrin and dieldrin on the biochemical mechanisms of the body have been the effects on hepatic microsomal enzymes. These changes occur in mammalian and avian species. The biochemical changes can usually be correlated with the histologic changes in the liver such as the proliferation of the smooth endoplasmic reticulum. These inducible enzymes while affecting the metabolism of a great many chemicals also metabolize steroids. Levels of aldrin and dieldrin as low as 1 ppm(150) cause changes in the metabolism of aminopyrine demethylase in the rat. The daily administration of 2 mg/kg dieldrin to female rats enhances the activity of aniline hydroxylase and p-nitroredutase(135). The administration of 3 mg/kg dieldrin enhances the hepatic metabolism of estradiol and estrone(264). Aldrin at a level of 20 ppm can affect the normal estrous cycle of rats without affecting the growth of the rat(8). The administration of 20 ppm dieldrin to hens increases aminopyrine demethylase activity and cytochrome P450 content(228). In the pigeon the feeding of 2 ppm dieldrin for 1 week increases the *in vitro* metabolism of testosterone and progesterone by liver microsomes(199). In combination with 10 ppm DDT there is an additive effect on the enhancement of testosterone and progesterone metabolism.

Liver mitochondrial activity is inhibited by the administration of 4 or 20 mg/kg dieldrin for 60 days. A depression NADH oxidase activity is seen(17). It is postulated that electron transport in the mitochondria is inhibited at the level of cytochrome b.

Xanthine oxidase and alkaline phosphatase of the livers of rats fed 50 or 100 ppm aldrin is decreased in activity(4).

The in vitro addition of aldrin and dieldrin causes a slight inhibition of the ATPases associated with oxidative phosphorylation and plasma membrane transport of cations(153). Mg^{+2} activated ATPase and the Na^{+}-K^{+} activated ATPase of membranes are inhibited by aldrin and dieldrin.

Long-term feeding of dieldrin to ducks affects behavior through a depression of amine levels in the brain(231). The administration of 30 ppm dieldrin to adult mallards and then immediately feeding the same level to their young for 70 to 76 days causes a depression of norepinephrine, dopamine and serotonin levels.

In rats fed a diet deficient in essential fatty acids, the addition of 20 or 30 ppm dieldrin to the diet will enhance the suppression of growth associated with essential fatty acid deficiency(250).

A ten day treatment of rats with 5 mg/kg dieldrin results in an increase in fasting blood glucose(126). A dose of 25 mg/kg given for 4 days will also increase the fasting blood glucose level by 60%. In these same animals the plasma corticosterone levels were 45 to 98% higher than controls. These authors conclude that dieldrin

has an effect on the normal utilization of endogenous glucose and also has an effect on the adrenal cortex.

It is reported that dieldrin given in a gelatin capsule at a dose of 20 mg/kg/day for 3 days to chickens causes a decrease in the uptake of labeled methionine by the heart sarcosomes and liver mitochondria in vitro(49).

The administration of dieldrin together with DDT leads to a decrease in the amount of DDT stored in the fat depots in rats and guinea pigs(256).

Using very low levels of dieldrin in dogs there is an indication that the prostatic fluid volume, the acid phosphatase activity of the prostatic fluid and the zinc content of the prostatic fluid is reduced. In these studies 15 ug/kg of dieldrin was given to dogs. The administration of 6-chloro-6-dehydro-17-acetoxy progesterone alone reduces these indices of prostatic fluid to a great extent. With the addition of dieldrin to the hormone there is less reduction of these indices, leading to the conclusion that there is a hormone-pesticide interaction(85).

APPENDIX III

CARCINOGENIC AND MUTAGENIC EFFECTS

Stephen S. Sternberg, M.D.

MAN

The largest single study of the effects of aldrin and dieldrin in man was by Jager (136). This was based on the occupational exposure of more than 800 workers at an insecticide plant over a period of about 15 years. From these workers, a group of 233 was selected for long-term exposure studies and consisted of personnel who had been exposed at least 4 years and up to 13-1/4 years, not only to aldrin and dieldrin, but to endrin, telodrin as well. The average blood level of dieldrin in this group was 0.035 ug/ml. This is equivalent to an average oral intake of 407 ug/man/day and represents over 50 times the daily intake of the general population of the U.S.A. Medical examinations of this group of workers revealed no adverse effects from this type of exposure. There were 23 instances of intoxication among aldrin/dieldrin workers during the 15 year study, none of which were fatal. Apparently, toxic manifestations were confined to the central nervous system, and they were reversible. In the long-term exposure group, there was no evidence of hepatic disease, nor was the liver enlarged on physical examination. In workers with high levels of dieldrin in the blood (at least 175 times the general population) no effect of pp'DDE metabolism was found; in those with 85 times that of the general population, no effect on steroid metabolism was seen, as measured by the ratio of

6-B-hydroxy-cortisol/17-hydroxy-corticosteroid excretion in the urine. The results of these determinations suggest a lack of enzyme induction in man.

Another noteworthy study was one in which volunteers ingested dieldrin (HEOD) on a daily basis, at a maximum dosage of 0.21 mg/man/day (132, 133). All medical tests performed at intervals and at the conclusion of the exposure at 24 months indicated a lack of effect. In particular, no liver abnormalities were found, nor were there any effects on the nervous system. Further, these same studies have shown that the levels of HEOD in the blood and adipose tissue are proportional to the daily dosage, and that there is a finite upper limit to storage which is also related to the daily intake and after equilibrum is reached. Thus, a steady-state occurs and there is no buildup within the body, and one can reasonably expect that in the absence of increased exposure, storage levels will not rise.

Long-term oral-feeding studies in animals

Primates

In a long-term feeding experiment with 31 Rhesus monkeys, animals were followed up to 6 years (270). Six were controls, and the remainder received various dosage levels of dieldrin in the diet. At levels below 1.75 ppm, no adverse effects were seen. Animals that received 1.75 ppm or more had ultrastructural hepatic changes and alterations in the intracellular hepatic enzymes, presumably related to hyperplasia of smooth endoplasmic reticulum, but no details are noted. In any event, the livers of all animals were otherwise normal, and no tumors were seen.

Dogs

In a 2-year feeding experiment with dogs (258) at dose levels of 0.005 and 0.05 mg/kg/day, no tumors were seen. There were no histopathologic alterations in the liver, although the alkaline phosphatase was elevated and the serum proteins slightly decreased in dogs that received the high dose.

In an earlier study (69) dogs were given doses at levels (2 to 10 mg/kg/day) which caused convulsions, fatty liver and bone marrow depression. The lowest dose used (0.2 mg/kg/day) was, in their study, a no-effect level, and dogs survived for as long as 25 months, the duration of the experiment. No tumors were seen.

Rats

A recent study with rats (Osborne-Wendel strain) fed up to 50 ppm of aldrin and dieldrin revealed no tumorigenic activity, but rather a lower overall tumor incidence in treated animals as compared to controls (54). A 2-year study (258) in CFE rats was made using dietary concentrations as high as 10 ppm. There was no increased incidence of tumors. However, 3 rats receiving the highest dose had microscope nodules in the liver; nodules were also present in one control rat. These are not further described.

Other studies have been performed in the rat which are difficult to interpret because of lack of pertinent information. In any event, there is no evidence that aldrin or dieldrin are carcinogenic in the rat.

Mouse

A number of long-term studies have been performed using mice of different strains. The most recent of these (259) indicated that

HEOD enhances the incidence of naturally occurring hepatic nodules in the CF1 strain. From the microscopic description of the 2 types of nodules, it appears that the so-called type (a) is benign and that the type (b) probably is a hepatocellular carcinoma. Both types are present in control animals of both sexes, but in general the type (a) is more frequent than the type (b). However, in the treated groups, the incidence of both types increases; to over 50% for type (b) mice treated with 20 ppm for 2 years, and as much as 77% for type (a) in mice at 5.0 ppm level. It is worth noting, however, that metasteses in mice with type (b) tumors were rare and in males were about the same as in control animals. Female mice in one experiment had a 4% incidence of lung metasteses of liver tumors, while no metasteses were seen in female controls. In another study, however, (52) in which Swiss mice were used, hyperplastic nodules were present in the treated animals, but no hepatic carcinomas. The control mice were free of nodules, quite unlike the CF1 mice used by Walker et al. (259)

Thus, the finding of an increased incidence of liver tumors in one strain of mice represents the closest approximation to what might be considered a carcinogenic effect of dieldrin.

The significance of an enhancement in the incidence of a naturally occurring tumor, as the only evidence of carcinogenicity, is difficult to interpret. Mice, in particular, have an unusual lability in response to various factors in long-term studies, and because of this are perhaps an unfortunate mammal to work with for carcinogenicity studies. It has been shown for instance that a "germ-free" status

in C3H mice in effect protected against the early development of liver tumors by a known carcinogen 7,12-dimethyl-benz(a) anthracene (213). Earlier Heston et al. (125) pointed-up the marked difference 2 standard diets had on the incidence of liver tumors in C3H mice, and Tannenbaum and Silverstone in 1949 (240), the variations produced by different levels of casein.

These and other considerations have led Roe and Grant (213) to suggest that some chemical agents which increase the incidence of certain tumors may act as co-carcinogens rather than carcinogens, and that in the absence of other evidence, a carcinogenic action should not be attributed to such agents.

In summary, no carcinogenic action has been demonstrated for dieldrin in rats, dogs or primates. In one strain of mice the CF1, an enhancement in the incidence of what are probably hepatocellular carcinomas was observed. The incidence of metasteses in male mice was the same in control and treated animals, while in treated female mice a relatively small percentage had metasteses. In another strain (Swiss) in which there were no malignant hepatic tumors in the controls, none were seen in the treated mice. If there is a carcinogenic action in dieldrin, it likely is a weak one at a level much like DDT.

Addendum

A major problem in evaluating long-term studies in animals concerns the description and classification of hepatic lesions. Not the least of these is use of the term "hepatoma." The word is used either for benign tumors or for malignant ones, but probably

most often as non-committal word when it is not known if the lesions are benign or malignant. In the latter instance, if no definition of the use of the word is given, and there is no satisfactory microscopic description and no photomicrographs, then the reader as well as the authors remain ignorant.

If there are difficulties in the pathological evaluation, these should be stated. Microscopic descriptions and representative photomicrographs often permit the reader to make his own judgement, and the value of the scienific work is greatly enhanced.

Non-mutagenicity of Aldrin/Dieldrin

In the literature review in "The Mutagenicity of Pesticides" by Epstein and Legator (66) there is a solitary reference to dieldrin (Markarian, 1966) wherein a 10% (sic) solution caused a "C-mitosis effect and no chromosome breaks" in sprouts of Crepis capillaris.

APPENDIX IV

RESIDUES IN THE ENVIRONMENT AND MAN

Richard A. Parker

A preliminary analysis of aldrin and dieldrin residues in the United States was conducted using a simulation model to develop a better understanding of the potential long-term levels in man. It must be emphasized that the specific results are of general utility only; formal optimization measured by goodness-of-fit to widely varying data in the literature was not attempted. Six components were incorporated: domestic aldrin and dieldrin used (1950-70 data from Shell Chemical Company), aldrin and dieldrin residuals in the environment, total intake by man from market basket food, and dieldrin content of human adipose tissue.

Persistence of these pesticides depends a great deal on soil type and climatic conditions. Estimates of half-life for dieldrin under field conditions range from two to seven years, considerably less for aldrin. Since an earlier simulation study (Parker unpublished) suggested that assumed half-lives of 1.4 to 3.5 years for aldrin and dieldrin combined had little immediate effect on the accumulation in man, figures given by Edwards (61) were accepted: 2.4 years for dieldrin and 0.51 years for aldrin. Equations adopted to approximate the quantities of aldrin (RA) and dieldrin (RD) present in the environment immediately after a particular annual application (A and D) were:

$$RA_t = A_t + RA_{t-1} \exp(-1.36)$$

$$RD_t = D_t + RD_{t-1} \exp(-0.29) + 1.27\, RA_{t-1} [\exp(-0.29) - \exp(-1.36)]$$

Aside from A_t and D_t, these equations form a solution to the following general pair of differential equations describing changes during one year:

$$\frac{dA}{dt} = -1.36A$$

$$\frac{dD}{dt} = 1.36A - 0.29D$$

Note that all of the aldrin was assumed to be converted to dieldrin.

It is well known that dieldrin is commonly found in meat, fish, poultry and dairy products (see, for example, Corneliussen 1970 (46) who gives values ranging from .004 to .052 mg/kg); residue levels may be assumed due largely to food intake rather than direct contact. Residuals in plant foods included in the diet of man are generally proportional to levels found in the soil on which the plants are grown; however, the proportionality constants are usually higher in oil containing crops like soybeans than in grain or root vegetables (39,111,152,170,200,267,269). Human dietary intake of aldrin and dieldrin has been studied extensively and summarized for five years by Duggan et al. (58):

	Year				
	1964-65	1965-66	1966-67	1967-68	1968-69
	ug/kg/day				
Aldrin	.01	.04	.01	.01	.001
Dieldrin	.08	.09	.05	.05	.07

The equation utilized to relate combined intake F with environmental residual was

$$F_t = 0.00088\ (RA_t + RD_t).$$

Since little data on human adipose levels is available before 1967, the constant was calculated to approximate the observed values for 1967-69. In addition, major modifications in analytical techniques make comparisons among many successive years questionable.

Dieldrin is usually present in human adipose tissue and, as expected, the concentration seems to be a function of geographical location (see the review by Jager 1970)(136). A continuing large scale sampling program is carried out in the United States by the Human Monitoring Survey, State Services Branch, Division of Pesticides, Community Studies, Environmental Protection Agency. The following survey results have been provided by EPA through Homer Wolfe, Chief of the Wenatchee Research Section:

Year	No. Samples	No. States	Method	Mean Conc (mg/kg)
1967	722	11	Non cleanup	0.14
1968	3,300	21	Non-cleanup	0.12
1968	3,237	23	Modified Mills (better cleanup)	0.13
1969	3,264	33 + Wash. D.C.	Modified Mills	0.13
1970 (inc)	2,626	27 + Wash. D.C.	Modified Mills	0.15

Jager (136) points out that De Jonge has estimated the half-life of dieldrin in man to be 0.73 years based on a study of 15 aldrin-dieldrin workers. Aldrin levels were apparently too low for detection, and no effort was made to distinguish aldrin and dieldrin in the simulation model. Change in human adipose tissue content T was described by the differential equation

$$\frac{dT}{dt} = 2.20F_{t-1} - 0.95T$$

Since much of the aldrin-dieldrin entering plant foods occurs during a relatively short growing season following pesticide application, the intake by humans was treated as constant ($2.20F_{t-1}$) during a particular year (0.95 corresponds to a half-life of 0.73 years).

The system of equations was solved for two cases: one in which aldrin and dieldrin were applied in 1971 and thereafter at constant annual rates of 8×10^6 pounds and 0.6×10^6 pounds, respectively, and another in which none was applied after 1971. The simulation output for both cases is presented in Figure 1. The first assumption yields predicted market basket intake of approximately 0.036 ug/kg/day in 1982 and concurrent adipose tissue content of 0.004 mg/kg. The second suggests 1982 levels of 0.002 ug/kg/day and 0.009 mg/kg.

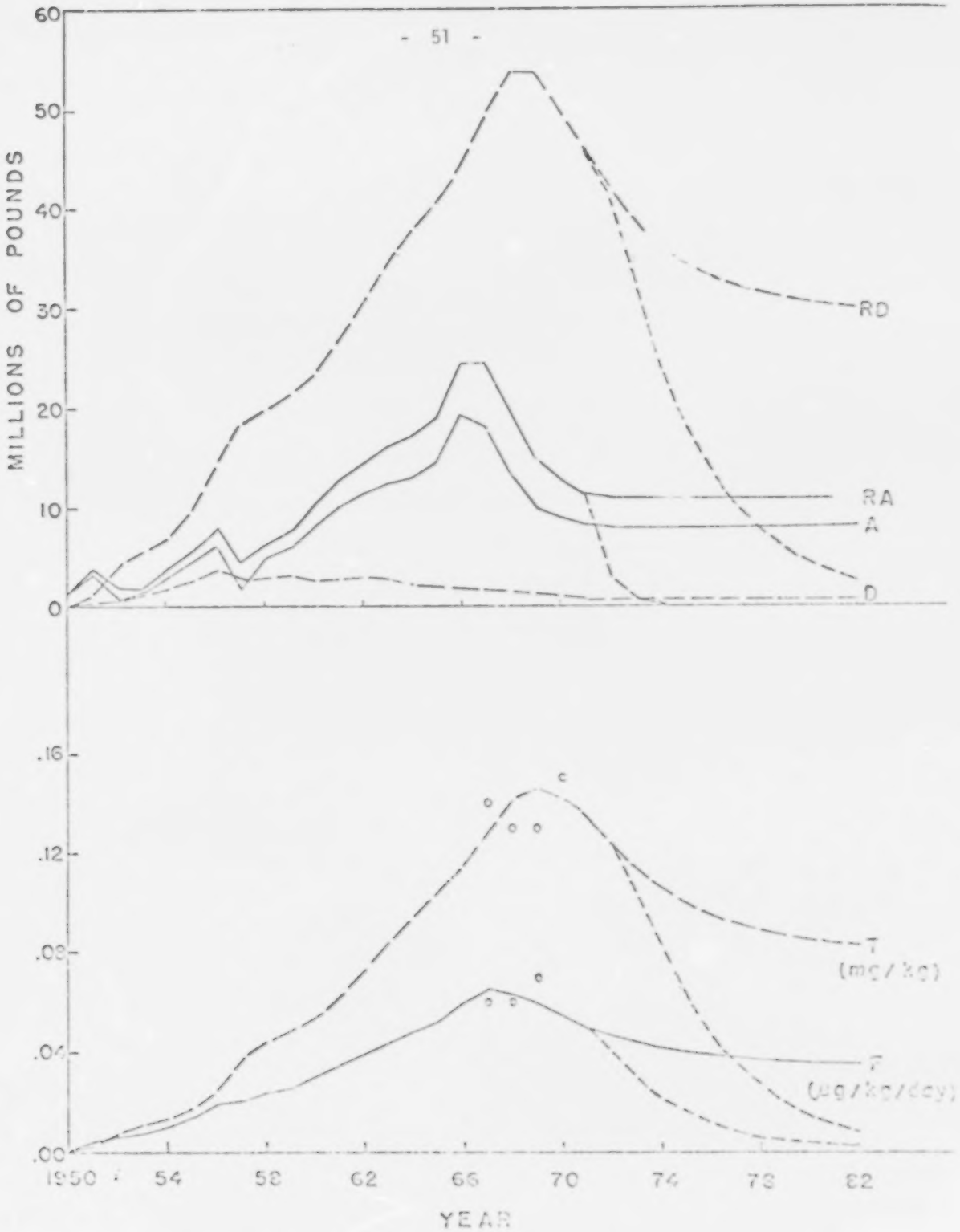


Figure 1. Simulation Results

APPENDIX V

ENVIRONMENTAL MOVEMENT AND CHANGE

Samuel D. Faust

Section I. "Occurrence, Distribution, and Persistence of Dieldrin and Aldrin in Air and Water"

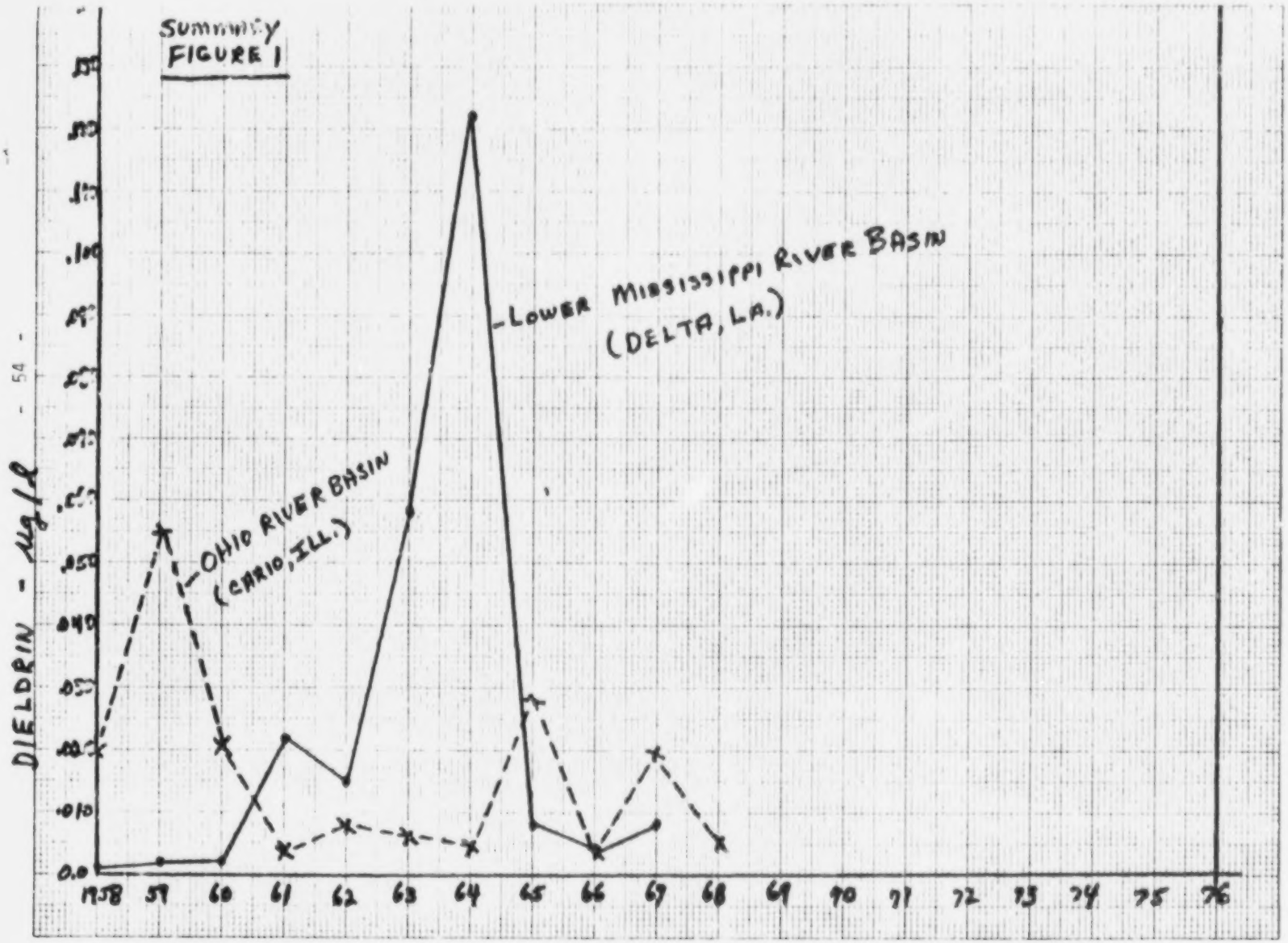
In view of the papers on the distribution, occurrence and persistence of dieldrin and aldrin in the atmosphere and various bodies of water, careful attention was given to the analytical techniques. It is extremely important that confirmation was made of the presence of these two pesticides. Most of the analysts used one form or another of gas-liquid chromatography with an electron capture detector. This detector is non-specific for organic pesticides, i.e., any organic compound that captures an electron and gives a response will be recorded. Therefore, there must be employed some such ancillary analytical technique as thin-layer chromatography or infrared spectroscopy that can identify at the molecular level. There does not appear to be any appreciable (see page 56) PCB interference with dieldrin and aldrin (as is the situation with DDT), but there are organic interferences in natural water, waste waters, bottom muds, stream sediments, etc.

Synoptic surveys of the major rivers of the U.S. have been conducted since 1958 by various agencies in the Federal Government (23,24, 25,139,163,219,261). Dieldrin appeared more frequently than, for example, endrin, DDT, DDE, DDD, BHC, etc., when placed on a basis of percent occurrence. Also, dieldrin appeared in almost all of the major U.S. rivers. Concentrations, however, were in the order of 10^{-6} to 10^{-9}

grams per liter (.001-1 ug/l). The highest concentration of dieldrin may have been .407 ug/l that was observed in the Tombigbee River at Columbus, Mississippi, in 1968. Aldrin was infrequently, if ever, reported. These surveys appear to be analytically sound and that it is certain that dieldrin was confirmed. These observed concentrations of dieldrin may be compared with the water quality criteria suggested by the EPA which are 10 ug/l for dieldrin, 10 ug/l for aldrin, and 10 ug/l for aldrin + dieldrin. These concentrations are being proposed now by the National Academy of Sciences to the EPA for adoption as water quality criteria and, as such, may undergo some revision before final acceptance. The concentrations of dieldrin and aldrin reported in the years 1958-1968 appear to be well below the proposed water quality criteria.

In order to determine a trend with time, dieldrin concentrations were plotted versus year in summary Figure 1 for two sampling sites. There does not appear to be any upward or downward trend in dieldrin concentration with time although the Ohio River Basin data may suggest a decreasing content. It should be remembered that these synoptic surveys grab samples at random. Therefore, these data may not be representative of the whole.

Some stream and river surveys were conducted in England for chlorinated hydrocarbons (47,130,176). In "clean" Scottish waters, dieldrin up to 0.01 ug/l was found. In "contaminated" streams, dieldrin concentrations >1 ug/l were found. In one survey (47), dieldrin was found in more than 50% of samples from various surface and ground waters in England and Wales. The highest concentrations of dieldrin were 2.48


Summary
FIGURE 1
DIELDRIN - ug/l
.100
.050
.040
.030
.020
.010
0.0
1958
59
60
61
62
63
64
65
66
67
68
69
70
71
72
73
74
75
76
OHIO RIVER BASIN
(CAIRO, ILL.)
LOWER MISSISSIPPI RIVER BASIN
(DELTA, LA.)

and .56 ug/l due to an industrial waste water discharge.

Some attempt has been made in the U.S. (43,237,263) and in England (1,2,242,266) to determine the extent of atmospheric transport of organic pesticides. Dieldrin, .003 ppm, was found in dust fallout collected in Cincinnati, Ohio (43,263). In an analysis of the atmosphere, air was sampled at nine locations in the U.S. (urban, rural, etc.). Dieldrin was found in only one location, Orlando, Florida, and in 50 of 99 samples. The maximum concentration was 29.7 ng/m^3. Aldrin was found in only one sample in only one location - Iowa City (8.0 ng/m^3). In both of these surveys no confirmation of the GLC peaks was attempted. Identification was made by relative retention time on two or three GLC columns. The English analysts were more careful than the U.S. analysts. They examined rainwater samples by thin-layer chromatography followed by gas-liquid chromatography and still called the results "apparent organo-chlorine insecticides" because not enough pesticide was available for infrared confirmation (265). Dieldrin concentrations in this survey ranged 9-28 ng/l. In another English rainwater survey (242), dieldrin contents of 1-40 pg/l (10^{-12} g/l) were observed in 7 sites throughout the British Isles. These concentrations represent a decrease from previous surveys due to "abandonment of large scale uses of aldrin and dieldrin" in Britain.

Some attempts have been made to evaluate the role of sediments in the transport of dieldrin and aldrin in surface waters (12,44). In a survey of the sediments of the lower Mississippi River, dieldrin concentrations ranged 0.15 to 931 ppm (dry weight) whereas aldrin concentrations ranged 1.09 to 567 ppm (12). These

sediments were collected from Wolf River and Cypress Creek, Memphis, Tennessee, near the manufacturer of endrin and heptachlor. Apparently, very little desorption of dieldrin and aldrin occurred because water samples showed .04-.37 ug/l dieldrin and 0.15-0.29 ug/l aldrin at the same sites.

In a persistence study (174), dieldrin, 1 mg/l, was added to a lake water and to a soil water obtained from percolation. After 7 months, .02 mg/l remained in the lake water and 0.16 mg/l remained in the soil water. After 12 months, 0.002 mg/l remained in the lake water whereas 0.07 mg/l remained in the soil water. This is very empirical and it is not clear on how the dieldrin disappeared.

Three papers were reviewed in order to answer the question of PCB interference with the gas-liquid chromatography of aldrin and dieldrin (205,206,208). It should be kept in mind that the most commonly used GLC columns for aldrin and dieldrin are the stationary phases of QF-1 and DC-200. Most of the interference data, however, were gathered from a 4% SE30 - 6% QF-1 column (205,206) where it was demonstrated that Aroclor 1254 with its 14 major peaks does, indeed, interfere with aldrin and dieldrin (i.e., the same relative retention times). However, I was able to find only one reference (208) where the DC-200 and QF-1 columns were used. In this one instance, dieldrin appeared as a trailing peak to one of the PCB peaks on QF-1 and came out ahead of the first major PCB peak on DC-200. No mention was made of aldrin.

Section II. "Dieldrin and Aldrin-Leaching from Soils - Sorption and Desorption"

There have been surprisingly few studies, field, or laboratory, that dealt specifically with the retention and release of aldrin and dieldrin in and from soils. From the meager information, aldrin and dieldrin appear to be retained in the soil with some downward and lateral movement (21,39,63,67,106,164,248). In a rice paddy study (164), no aldrin or dieldrin was found 5" from the drill row in which aldrin was applied. Very low concentrations of aldrin and dieldrin were found in the rice paddy water (<.008 ug/l aldrin and .17 ug/l dieldrin). In fact, these concentrations were about the same as for an untreated area. In an adsorption on soil study (67) there was somewhat of direct relation between the amount of dieldrin adsorption and organic matter content of soil. As the percent organic content was increased, the amount if dieldrin, ng/g, was increased. There was some evidence presented also (236) that more dieldrin may be lost (as measured by vapor density) from "wet" soils than from "dry" soils. Apparently, water molecules may replace previously adsorbed dieldrin and when the soil becomes saturated with water, the vapor density of dieldrin becomes constant and independent of soil water content. In a translocation study (63,248), dieldrin was applied to soils adjacent to four small ponds under three conditions: no incorporation in the soil, light incorporation, and thorough incorporation. In each case, no dieldrin (below analytical sensitivity) was found in the pond water, and very little in pond mud (< .02 ppm to .13 ppm, for example). Most of the dieldrin was

found in the soil where it was originally applied.

Section III. "Aldrin and Dieldrin Residues in Soils, Persistence, Degradation, Etc."

Many field and laboratory studies have been performed on the occurrence, persistence, etc., of aldrin and dieldrin in the soil environment. In the case of persistence (51,166,167,168,169,170,171, 172,173,192), aldrin and dieldrin disappeared slowly from soils, for example, aldrin, 15.6 ppm, "decayed" to .860 ppm dieldrin over a 5-year period (169,170). In a field study (266), aldrin and dieldrin were observed to disappear in accord with first order kinetics (aldrin $\longrightarrow$ dieldrin, then decay of the dieldrin), whereupon a "half-life" of 2-4 years for dieldrin was calculated. In another field study (192) aldrin was found to have a half-life value of 5 years and dieldrin of 7 years. In still another field study (173), it was found that the presence of a cover crop permitted a greater persistence of aldrin and dieldrin than when these pesticides were incorporated into soils or applied on the soil surface. This study (173) speculated that the greatest loss of aldrin and dieldrin from soils is through volatilization. Four months after application of aldrin to the soil's surface (173), most all of the aldrin and dieldrin were found in the upper 2" and 75% in the upper 1". Twelve months after application, 90% was found in the upper 3". Three years after application, 72-82% was found in the upper 3", 11-16% was found in 3-4", and "very little" was found in the 4-9" depth. It should be stated here that many of these studies used application rates of aldrin and dieldrin (5-25 lbs/ acre) that are far in excess of actual agricultural application rates

(1,3,4, lbs/acre). There have been some analytical surveys of fields treated with aldrin and dieldrin (57,108,265) where these pesticides were found several years after initial treatment. One significant study (81), however, should be mentioned. Soil samples, stored since 1909, were analyzed for chlorinated hydrocarbons under conditions commonly employed in soil surveys. 32 of 34 samples showed "apparent" insecticide residues! In fact, "aldrin" was frequently observed. Epoxidation of aldrin to dieldrin was observed (168) which, by now, should be common knowledge. Some vegetable crops were grown on aldrin treated soils (25 lbs/acre). Translocation was observed with carrots taking up the greatest amounts of aldrin (up to 1.5 ppm). However, when the carrots were grown on normal dosages of aldrin (1 lb/acre), .05 -.09 ppm were observed in this crop. Potatoes, beets, radishes, cucumbers, lettuce, and turnips took up considerably lesser quantities of aldrin and dieldrin.

Soil type is an apparent factor also in the persistence of aldrin and dieldrin (12,172). Under laboratory studies, aldrin (applied 200 lbs/6" acre, 26°C), disappeared faster from Plainfield sand (0.8% organics) than from Miami silt loam (3.8% organics) than from muck soil (40% organics) observed over 56 days. Apparently, the organic matter retards whatever mechanism is responsible for the aldrin disappearance (note that the word degradation is not used). Similar results were observed under laboratory conditions. Temperature was observed also to be a factor as aldrin (20 lbs/6" acre) disappeared faster from a silt loam soil faster at 46°C than at 26°C than at 6°C (laboratory study over 56 days). In some early soil persistence studies (166,171), aldrin would disappear to become undetected chemically but a "toxicant"

to flys would remain behind. This "toxicant" proved to be dieldrin.

Section IV. "Microbiological Degradation of Aldrin and Dieldrin"

Very little research has been reported on the microbiological degradation of aldrin and dieldrin. The most probable reason may be that these pesticides are not degradable, at least, in accord with this reviewer's definition (a change in the oxidation number of carbon to +IV, i.e. CO_2). What information available shows that (a) dieldrin may be converted to water soluble and solvent soluble metabolites by soil organisms (179,180), (b) dieldrin may be converted to photodieldrin by microbial isolates from various environments (181), (c) conversion of aldrin to dieldrin by a soil fungus (251), (d) partial hydrolysis of dieldrin by aerobacter aerogenes (262), and (e) partial degradation of dieldrin to CO_2 by a soil fungus (18). It appears that a wide variety of soil organisms can muster their courage to attack aldrin and dieldrin, but that most give up since metabolites are formed which have the basic ring structure in tact. There was one hardy soil fungus Trichoderma Koningi that thinks he can break the ring structure as CO_2^{14} was produced from C^{14} dieldrin.

Section V. "Photodecomposition of Aldrin and Dieldrin"

It appears that aldrin and dieldrin in solid states or in solution may be converted by ultraviolet light to other products which are usually more toxic to flies, mosquitoes, mice, etc., than the parent compounds. The central question is, however, can sunlight catalyze the decomposition of aldrin and dieldrin? The photoconversion of dieldrin is wavelength dependent, i.e., it requires short-wavelength, high energy irradiation of 2600A° and lower. The earth's atmosphere

absorbs all light of wavelengths less than 2863A°. Experiments were conducted (122) in an effort to answer the photoconversion of dieldrin under natural solar conditions. The leaves of corn plants were sprayed with acetone solutions of dieldrin, dried, and placed in the beautiful California sun for 64 days. Also some dieldrin sprayed corn leaves were irradiated in the laboratory. Here the results as presented in the paper become confusing. These statements were made without the supporting evidence: "Gas chromatographic analysis of the residue remaining on corn plants which had been treated with dieldrin showed that none of the pentachloro compound (IV) was formed. Instead another substance appeared which was identified as the photoisomerization product (III) by comparison with an authentic specimen. However, when the dieldrin-treated leaves were exposed to 2537A° ultraviolet light in the laboratory, considerable amounts of IV were found after only 15 minutes of irradiation." Based upon experiments, the authors stated "this particular reaction (dieldrin to compound IV when a H replaces one of the vinyl chlorines) would not be expected to occur in the field during exposure to natural sunlight." Furthermore, compound III could not be detected, in the laboratory, after irradiation of dieldrin solutions at wavelengths between 2500 and 3000A°. The significance of compound III is that it is "approximately two times more toxic than dieldrin to the house fly and mosquito," (216).

APPENDIX VI

SOIL INSECT CONTROL

Mahlon L. Fairchild and R. L. Doutt

Wireworms

Scott and Carpenter (221) studied wireworm control on potatoes in Idaho with side-dressed and broadcast insecticide applications. In one experiment Bay-38156 and disulfoton were side-dressed at rates of 8 and 18 ozs. A.I./1000 ft. row. Agritox® and Dasanit® were side-dressed at .8 and 1.8 oz. A.I./1000 ft. of row. Propoxur was side-dressed at .9 and 1.7 oz. A.I./1000 ft. of row. All treatments resulted in significant reductions in wireworm injury. In a second test, chlordane was broadcast at the rate of 10 lbs. A.I./acre; diazinon and parathion at 6 lb. A.I./acre; phoxim at 3 lbs. A.I./acre, and Dyfonate® and Agritox® at 4 lbs. A.I./acre. Phorate was side-dressed at 2.3 oz. A.I./1000 ft. of row and dichlorous at 3.8 oz. A.I./1000 ft. of row. The results indicated that 2 organophosphates compounds control wireworms as well as chlorinated hydrocarbonous materials, represented by chlordane. These 2 materials were: Dyfonate® and Agritox®. Diazinon, phorate, and parathion gave poor control in these tests. Dichlorous and phoxim were intermediate in the control. Results of this experiment were not very promising. Even if 80% control could be achieved in a population which could cause 20% damage, the remaining wireworms could cause 6% damage. This level of control would not be feasible economically under the present grading system, since the potatoes would not meet U.S. No. 1

grade requirements. Further 80% control would still allow many wireworms to mature, mate, and lay eggs.

Keaster and Fairchild (143) studied control of sand wireworms in Missouri. American Cyanamid 18133 at 1.2 lbs. A.I./acre, Heptachlor at 1.5 lbs. A.I./acre and Hercules 5727 at 1.13 lbs. A.I./acre, seemed to give significant reduction in wireworm injury to corn.

Tappan (241) tested 17 insecticidal compounds in various formulations for wireworm control on cigar-wrapper tobacco. Compounds which gave good results were: Bayer 25141, diazinon, GS-13005, Kepone, N-290, Niagara 9203 and parathion.

Onsager et al. (198) investigated control of wireworms on potatoes in eastern Washington by soil fumigants and organophosphorous insecticides. These investigators found that Telone®, DD® Mixture, and ethylene dibromide applied at 20 gal., 25 gal., and 36 lb./acre, respectively, and broadcast applications of granulated diazinon, parathion, and phorate, applied at 2, 4, and 3 lbs./acre, respectively, gave economic control of the Pacific Coast wireworm on potatoes in sandy loam soil. Cullage was reduced 93.6-100%. Granulated disulfoton, broadcast at 3 lb./acre was reported to give significantly less control. Post-planting side-dressings of granulated parathion, phorate, Stauffer N-2790, and Bayer 25141, applied at 3 lb./acre reduced cullage 74.4-85.2%. Granulated disulfoton and Bayer 37289 and naphalene crystals were significantly inferior. Phorate and disulfoton granules gave as good control when metered onto the soil surface in bands 6 in. wide in the furrow left by the press-wheel potato planter as when side-dressed in the soil at

the same rate. Of all diazinon, disulfoton, parathion, and phorate treatments only phorate applied in the press-wheel furrow left a detectable residue above the tolerance in tubes collected 3 weeks before harvest.

Harris (1965) reported diazinon baits, Kepone baits, parathion granules and diazinon granules to be somewhat effective against wireworms in sweet corn. These studies were conducted in organic soils in Florida.

Brett et al. (1966) reported that diazinon gave poor control of wireworms in sweet potatoes when it was incorporated into the soil prior to planting, but a foliage-surface application during August gave good control. Diazinon gave good control when applied on the surface late in July at the rate of 3 lb. active ingredient per acre in a granular formulation. Application in August were less effective. Insecticides incorporated in the soil were less effective in organic soils than in sandy soils. Soil type show little effect on control by surface-applied materials.

Burrage et al. (31) reported that granules of N-2790 and Zenophos at 5 lb./acre broadcast and worked into the soil shortly before planting, gave more than 90% wireworm-damage-free potatoes compared with 26 to 55% damage-free potatoes in untreated soils. Diazinon at 7 lbs./acre and Bay 25151, and Bay 37289 and UC 10854 at 10 lbs./acres also gave more than 90% damage-free potatoes. Lower rates tested gave less than the 90% damage-free level required for Canada No. 1 potatoes. Granules of Bay 37289 and N-2790 broadcast at 10 lbs./acre caused an objectionable flavor.

In 1969, Onsager (196) reported that fumigation of the soil in autumn with ethylene dibromide, Telone® or dichloropropane-dichlorapropene mixture gave excellent control of Pacific Coast wireworms. Dyfonate at 2 lbs./acre, 3/4 lb./acre of diazinon or parathion, and 4 lbs./acre of carbofuran or Bay 37289 also gave excellent control when broadcast. Broadcast treatments tended to be more effective and less variable than band or side-dress treatments with the same chemicals. Band treatments of 4 lbs./acre of granular disulfoton and broadcast treatments with up to 6 lb./acre of granular Shell SD 8530 were inferior to other treatments. However, Onsager and Foiles (197) reported when granular carbofuran, Dyfonate®, and parathion were applied in mid-March for control of the Great Basin wireworm, and the Pacific Coast wireworms on summer potatoes that side-dressed treatments at a rate of about 2.3 lb. of toxicant per acre gave better average control with less variation than broadcast treatments with 4-8 lb. of the same toxicant per acre.

Flooding infested fields for 6 to 7 days with an inch or two of water during extremely hot weather has been reported to kill nearly all wireworms. Soil temperature must remain above 68°F. for the entire period for best results. In contrast, when the upper 15 inches of infested soil is allowed to become very dry for several weeks in summer, most of the wireworms, especially the younger ones, are killed. Drying has been recommended to keep wireworms below economic levels on irrigated lands.

Plowing fields in the summer during the pupal stage has been reported to reduce wireworm numbers. Mechanical injury to the worms and exposure to summer heat and low humidities account for most of the mortality at this stage.

Crop rotation in certain areas have been reported to reduce wireworms. The growing of truck crops in the same area continuously will usually cause a build-up of wireworms. The growing of potatoes in a short rotation with clover or grain is undesirable because of wireworm build-up (160).

Cutworms

Morgan and French (188) evaluated fifteen compounds for control of granulate cutworms on peanuts in Georgia. The materials were used as baits, sprays, and one dust mixture in experiments during a 4-year period 1966-69. Abate®, monocrotophos, Dursban®, Dyfonate®, trichlorfon, methomyl, and Monitor® gave higher than 90% control of cutworm larvae within 24 hours after application. In general the mixtures containing wheat bran gave the highest percent control.

Harris et al. (1969) evaluated four insecticides for control of the dark-sided cutworm in rye grown in rotation with tobacco. The four materials tested were DDT, Bay 37289, Dursban, and trichlorfon. In plots using artificial infestations DDT and Dursban gave 100% control while Bay 37289 provided 90% control, trichlorfon at 1 lb. per acre was not sufficiently effective. It was noted that DDT, Dursban and Bay 37289 provided 88-90% control of heavy natural infestations. It was noted, however, that the rapid dissipation of the residues of Dursban and Bay 37289 would require a split application to give

adequate control.

Harris and Svec (113) studied the effects of several insecticides on black cutworm in the laboratory. These studies indicated that Dursban, Bayer 37289, and parathion warrant further investigation as possible alternatives for the cyclodiene insecticides as soil treatments for control of the black cutworm. It was noted, however, these materials should be applied when the larvae are in the early instars to be effective. Soil type, moisture, method of application, and formulation must be taken into consideration for effective control.

Further studies by Harris and Svec (115) showed three other compounds having potential as possible replacement for cyclodiene compounds as control agents for dark-sided cutworms. These compounds were: N-2596, phoxim and chlorphoxim. Chlorphoxim seemed to be somewhat less effective than the other two compounds.

Unpublished studies by Sechriest and York in 1966 (227) indicated that Niagara 10242 (Furadan®), and a combination of thimet and A.C. 47470 gave effective control against black cutworms. These studies were conducted using artifical infestations. In a further unpublished screening test conducted by Sechriest in 1966, Dursban®, Sevin®, A.C. 47470 + Thimet® and G.S. - 10133 as well as Baygon® showed promise as effective insecticides against black cutworms.

Sechriest (224) reported that Baygon® did not perform well as a cutworm insecticide. However, diazinon, Dyfonate®, dylox, Sevin®, Bay 37289, GC-6506, EI-52160, and Thimet + EI-4740 were shown to be effective against cutworms.

Begg et al. in 1963 (15) reported that soil treatments in general did not reduce the infestations of the black cutworm to non-economic levels during the first seven days after treatment. It apparently took some time for the larvae to contact a lethal amount of an insecticide in the soil. The results of field test conducted with eight insecticides indicated their relative effectiveness in test could be rated endrin>aldrin = dieldrin = heptachlor>DDT = Dylox>Guthion = Sevin.

According to work done by Harris and Mazurek in 1961 (110), the regression line slopes would indicate that a small increase in the rate of application of dieldrin should result in more effective control of cutworms. Conversely, large increases in rate of applications of Dylox, Guthion, and Sevin would result in little or no increase in insecticide efficiency.

Laboratory studies by Harris and Gore in 1971 (109) were conducted to determine the toxicity of 3 insecticides, DDT, Dursban®, and Galecron®, to the various stages in the life cycle of the dark-sided cutworm. The results indicate that DDT was effective against the early larval stages, but ineffective against the later larval stages, eggs, pupae, and adults. Dursban was effective against eggs, early larval stages, and adults, but ineffective against the later larval stages and pupae. Galecron was highly selective and was effective against eggs.

Laboratory tests by Harris and Svec in 1968 (114) indicated that 5 of 7 insecticides tested as direct-contact poisons against 3rd- and 4th-instar variegated cutworms were more effective than aldrin.

The most toxic was Lannate® >DDT>parathion>Dursban® >Ciba 8874. Birlane® and Bayer 37289 were slightly less toxic than aldrin. As soil surface applications, Dursban and aldrin were highly effective, DDT, parathion and Bayer 37289 were only slightly less so, and Lannate was ineffective. Soil-surface applications of Dursban were 2-4 times as effective as soil incorporations. Dursban, DDT, and Lannate were all highly effective as stomach poisons. Lannate was 32 times more effective as a stomach poison than as a soil insecticide.

Greenhouse studies conducted by Sechriest (225) indicated several baits to be effective against black cutworm populations. The baits were: Trichlorfon Bait C, CL-47470 - 2% apple pomace, Abate - 2% apple pomace, ethyl parathion - 2% apple pomace, mirex - 1.25% bran, TDE - 5% bran, and carbaryl - 5% bran. Apple pomace and wheat bran were effective baits and the placement within 6 in. of the row resulted in satisfactory kill of larvae.

Studies by Harris et al. in 1962 indicated that there was very little resistance building in black cutworms in certain areas of Canada to soil insecticides. These studies also indicated that in general organophosphates were less effective against black cutworms than cyclodiene insecticides.

White Grubs

Several methods have been suggested for controlling white grubs. Fleming and Baker (73) suggested using hot water to control the Japanese beetle in the soil, on the roots of nursery plants. Metzger and Maines in 1935 (182) as well as Fleming et al. (79)

reported on the success of using traps baited with geraniol and eugenol for control of white grubs, particularly the Japanese beetle.

The effectiveness of lead arsenate to control white grubs has been explored by researchers such as Fleming and Baker (74), Fleming et al. (78) and Fleming (70). Napthalene and Derris have been reported to be helpful repellents of the Japanese beetle by Fleming and Baker (75, 76).

Many studies have been conducted using cyclodienes to control grubs. Fleming in 1947 reported that DDT was very effective against the grub of the Japanese beetle. Hadley and Fleming (101) reported on the use of other cyclodiene compounds such as TDE, chlordane, methoxychlor as well as ethylene oxide, methyl bromide, lead arsenate and several imported parasites for control of the larvae of the Japanese beetle. Fleming and Baker (77) also reported that paris green and its homologues were effective insecticides against Japanese beetle larvae.

Burrage and Gyrisco (29,30) studied the control of the European chafer in pasture sod. The test conducted by Burrage and Gyrisco included six materials - parathion, dieldrin, aldrin, chlordane, BHC, and DDT; parathion and BHC did not perform as well as the other materials. Grambell (91, 92, 93, 94, 95, 96, 97, 98) did extensive work on controlling the European chafer. Most of these works included the use of cyclodiene insecticides for control. Tashiro (243) reported that methyl bromide fumigation gave satisfactory control of the European chafer in nursery stock.

Hallock (102) reported that carbon-disulphide emulsion as well as lead arsenate could be used to control Asiatic beetle larvae in lawns.

Several parasites have been reported to attack Scarabaeidae larvae. Gardner and Parker (83), Glaser (86), Fleming (77), Burrel (32), King and Holloway (148) and Dutky and Gooden (60) are among the researchers who have studied parasites of the white grubs. Milky diseases in beetles have to be reported to give rather effective control of several white grubs. Hawley (119) and Fleming (1933) among others conducted studies which show the effectiveness of milky diseases.

APPENDIX VI - A

OCCURRENCE OF ALDRIN AND DIELDRIN IN BIOLOGICAL ENVIRONMENTS

Samuel D. Faust

Dieldrin Contents of Bottom Feeding Fish

Four papers were reviewed in connection with the dieldrin contents of bottom feeding fish. Dieldrin was found in carp, suckers, channel catfish, and bigmouth buffaloes. In general, the contents were less than 1 ppm (usually, whole fish). Also, the dieldrin contents were less than DDT contents (see Table in ref. 121). The analytical methodology consisted of the usual solvent extraction, cleanup on Florisil and gas-liquid chromatographic separation on DC-200 or some other non-polar phase with an electron capture detector. Very, very seldom was any confirmatory technique applied. An occasional thin-layer chromatogram was tossed in. Only one paper (121) found (or at least reported) aldrin in these fish. Another general observation may be made: the bottom feeders did not appear to have dieldrin contents any higher than the non-bottom feeders. For example, two small mouth bass (a bottom feeder) caught in the Potomac River showed dieldrin contents of .05 and .01 ppm whereas two large mouth bass showed dieldrin contents of .03 and .06 ppm (121).

APPENDIX VI - B

OCCURRENCE OF ALDRIN AND DIELDRIN IN BIOLOGICAL ENVIRONMENTS

Samuel D. Faust

Marine Environments

A few papers were reviewed in order to gain some insight into the occurrence of aldrin and dieldrin in marine environments, especially the estuary. There is some evidence that oysters, mussels, crabs, etc., contain residues of aldrin and dieldrin (28, 154, 187, 218). However, the frequency of occurrence and the concentrations were not as great as suspected. For example, "dieldrin was not routinely found" in oysters where typical concentrations were 10, 27, 20, 11, 18 ppb (ug/kg) (187). By routinely found may be interpreted as the occurrence of dieldrin in less than one-half of all samples. An interesting observation came from the California study (187): "expected high levels of pesticides were not found in San Francisco Bay, the terminating point for the Sacramento and San Joaquin Rivers which drain over 6 million acres of agricultural land in the Sacramento and San Joaquin Valleys." Aldrin was reported in 17 of 133 samples of oysters taken from South Atlantic and Gulf of Mexico waters at concentrations of 0.01 to 0.03 ppm with 0.01 ppm as the median.

APPENDIX VII

IMPACT OF WITHDRAWAL OF SOIL USES OF ALDRIN AND DIELDRIN

Mahlon L. Fairchild

Very little valid information is available indicating the economic impact resulting from the withdrawal of aldrin and dieldrin. Various individuals have attempted to use the meager information on soil insect losses to predict a dollar value that might be lost if soil insecticides were not used or substitutes were used in place of the cyclodiene insecticides. Estimates of possible losses range from $50,000,000 to $150,000,000 for corn alone. Of course additional insect losses would be suffered on the many other commodities where cyclodiene compounds are used for control of soil inhabiting arthropods. Although these losses at first appear staggering this may not place an immediate limitation on meeting the needs of corn production for our nation. However, the withdrawal of these materials may result in a complete loss of an individual farmer's crop.

Unfortunately soil insecticides are used as a preventive to soil insect damage. If an individual farmer lost one crop every three years it would result in economic disaster to the individual farmer but may not have a tremendous impact on the corn production in the nation.

It is embarrassing to entomologists to admit how little they know about the soil inhabiting arthropods. Before a preventive method of insect control can be eliminated one must have the ability to predict insect outbreaks. Unfortunately at the present time farmers do not even know the number of various soil inhabitating pests they are

controlling but are positive of return of their investment whenever a pesticide is used. Furthermore if the pest is known and can be identified in the field very little is known about the life history, habits or host ranges of the many soil pests. The soil insect complex is made up of white grubs, wireworms, cutworms, seed beetles, maggots, and other incidental problems and little is known about individual species within each group.

Another point that should be mentioned is the drastic changes that have taken place in agricultural production in the last 20 years that are coincidental with the use of soil insecticides. It has been suggested that pesticides were not necessary 20 years ago and, therefore, should not be necessary today. One must remember that changes in fertility, irrigation procedures, plant stands all relate to the economics of crop production and are also interrelated with the use of pesticides. To illustrate the importance of these changes the average per acre corn yields in 1950, 1960, 1970, and 1971 were 38.2, 54.7, 71.6, and 86.8 bushels, respectively. This represents over a 2-fold increase and undoubtedly makes the insect control more essential. Before one can back up 20 years in pesticide usage, consideration should be given to the impact this would have in interrelationships with all of the other factors that have changed. Intensified agriculture has actually moved some pests from the relatively unimportant to economic problems when the above-mentioned changes took place.

RESEARCH NEEDS

Additional research should be initiated to establish economic thresholds for the various soil inhabiting arthropods. Additional work should also be undertaken to study the distribution, abundance, biology and ecology of soil inhabiting arthropods. All of the above information is basic to an integrated pest management approach to insect control. This method of control includes non-chemical (biological, cultural, host plant resistance, etc.) and limited chemical usage.

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PERSONS APPEARING BEFORE THE COMMITTEE

First Meeting

December 16, 1971

Mr. Harold G. Alford, Pesticides Regulation Division, Environmental Protection Agency

Mr. Thomas H. Kemp, Office of General Counsel, Environmental Protection Agency

Second Meeting

January 25 & 26, 1972

Mr. Walter Appleby, Shell Chemical Company

Mr. William Bernholz, Drew Chemical Company

Dr. John Berry, Economic Research Service, United States Department of Agriculture

Mr. Piet Bodenhorst, Lees Carpets, Div. of Burlington Industries

Dr. Frederick Coulston, Albany Medical College of Union University

Mr. Walter W. Goeppinger, National Corn Growers Association

Dr. Ralph Heal, National Pest Control Association

Dr. John McPhee, International Wool Secretariat

Mr. Lou Mitchell, Shell Chemical Company

Dr. Paul Porter, Shell Chemical Company

Mr. John Redston, Drew Chemical Company

Mr. G. Gregory Rohwer, Animal and Plant Health Service, United States Department of Agriculture

Dr. Joseph Sloan, Shell Chemical Company

Dr. M. B. Slomka, Shell Chemical Company

Mr. Robert Thatcher, Forest Service, United States Department of Agriculture

Dr. Fred Tschirley, Office of Secretary, United States Department of Agriculture

Dr. H. G. S. Van Raalte, Shell Chemical Company

Dr. Charles F. Wurster, Environmental Defense Fund

Third Meeting

February 23, 1972

Dr. F. F. Becker, College of Medicine, New York University

Dr. J. H. Berry, Economic Research Service, United States Department of Agriculture

Dr. D. L. Coudriet, Agricultural Research Service, United States Department of Agriculture

Dr. P. J. Luginbill, Agriculture Research Service, United States Department of Agriculture

Dr. P. H. Schwartz, Agricultural Research Service, United States Department of Agriculture

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